EBI Dbfetch

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SV   L01624.1
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DT   23-MAR-1993 (Rel. 35, Created)
DT   09-SEP-2004 (Rel. 81, Last updated, Version 4)
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DE   Rattus norvegicus serum and glucocorticoid-regulated kinase (sgk) mRNA,
DE   complete cds.
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KW   serine/threonine kinase; serine/threonine protein kinase.
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OS   Rattus norvegicus (Norway rat)
OC   Eukaryota; Metazoa; Chordata; Craniata; Vertebrata; Euteleostomi; Mammalia;
OC   Eutheria; Rodentia; Sciurognathi; Muridae; Murinae; Rattus.
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RN   [1]
RP   1-2435
RX   MEDLINE; 93204949.
RX   PUBMED; 8455596.
RA   Webster M.K., Goya L., Ge Y., Maiyar A.C., Firestone G.L.;
RT   "Characterization of sgk, a novel member of the serine/threonine protein
RT   kinase gene family which is transcriptionally induced by glucocorticoids
RT   and serum";
RL   Mol. Cell. Biol. 13(4):2031-2040(1993).
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Search Results - Record(s) 1 through 5 of 5 returned.

☐ 1. Document ID: US 20030077827 A1

Using default format because multiple data bases are involved.

L3: Entry 1 of 5 File: PGPB Apr 24, 2003

PGPUB-DOCUMENT-NUMBER: 20030077827
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20030077827 A1

TITLE: Surface transfection and expression procedure

PUBLICATION-DATE: April 24, 2003

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Uhler, Michael D.	Ann Arbor	MI	US	

US-CL-CURRENT: 435/455; 435/325, 435/6, 435/69.1

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 2. Document ID: US 20020197720 A1

L3: Entry 2 of 5 File: PGPB Dec 26, 2002

PGPUB-DOCUMENT-NUMBER: 20020197720
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020197720 A1

TITLE: Surface transfection and expression procedure

PUBLICATION-DATE: December 26, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Uhler, Michael D.	Ann Arbor	MI	US	

US-CL-CURRENT: 435/458; 435/325

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw De

☐ 3. Document ID: US 20020146825 A1

L3: Entry 3 of 5 File: PGPB Oct 10, 2002

PGPUB-DOCUMENT-NUMBER: 20020146825
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020146825 A1

TITLE: Surface transfection and expression procedure

PUBLICATION-DATE: October 10, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Uhler, Michael D.	Ann Arbor	MI	US	

US-CL-CURRENT: 435/455; 435/458

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 4. Document ID: US 20020123056 A1

L3: Entry 4 of 5 File: PGPB Sep 5, 2002

PGPUB-DOCUMENT-NUMBER: 20020123056
PGPUB-FILING-TYPE: new
DOCUMENT-IDENTIFIER: US 20020123056 A1

TITLE: SGK2 and its uses

PUBLICATION-DATE: September 5, 2002

INVENTOR-INFORMATION:

NAME	CITY	STATE	COUNTRY	RULE-47
Delaney, Allen	Vancouver		CA	
Yoganathan, Thillainathan	Richmond		CA	

US-CL-CURRENT: 435/6; 435/7.23, 536/23.2, 800/3

Full | Title | Citation | Front | Review | Classification | Date | Reference | Sequences | Attachments | Claims | KWIC | Draw D

☐ 5. Document ID: JP 2002533063 W, WO 200035946 A1, EP 1141003 A1

L3: Entry 5 of 5 File: DWPI Oct 8, 2002

DERWENT-ACC-NO: 2000-442364
DERWENT-WEEK: 200281

TITLE: Modulation of serum and glucocorticoid-induced protein kinase (SGK) by phosphorylation with 3-phosphoinositide-dependent protein kinase-1 (PDK1) or dephosphorylation, useful for treatment of cancer, diabetes and ischemic diseases

INVENTOR: COHEN, P; DEAK, M ; KOBAYASHI, T

PRIORITY-DATA: 1999GB-0019676 (August 19, 1999), 1998US-112217P (December 14, 1998)

PATENT-FAMILY:

PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
JP 2002533063 W	October 8, 2002		125	C12N015/09
WO 200035946 A1	June 22, 2000	E	127	C07K014/435
EP 1141003 A1	October 10, 2001	E	000	C07K014/435

INT-CL (IPC): A61 K 45/00; A61 P 3/10; A61 P 9/10; A61 P 35/00; A61 P 43/00; C07 K 14/435; C07 K 19/00; C12 N 1/15; C12 N 1/19; C12 N 1/21; C12 N 5/10; C12 N 9/12; C12 N 15/09; C12 N 15/54; C12 N 15/63; C12 P 21/02; C12 Q 1/48; G01 N 33/15; G01 N 33/50

Terms	Documents
serum and glucocorticoid-induced protein kinase	5

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L6: Entry 1 of 1 File: DWPI Oct 8, 2002

DERWENT-ACC-NO: 2000-442364
DERWENT-WEEK: 200281

TITLE: Modulation of serum and glucocorticoid-induced protein kinase (SGK) by phosphorylation with 3-phosphoinositide-dependent protein kinase-1 (PDK1) or dephosphorylation, useful for treatment of cancer, diabetes and ischemic diseases

INVENTOR: COHEN, P; DEAK, M ; KOBAYASHI, T

PRIORITY-DATA: 1999GB-0019676 (August 19, 1999), 1998US-112217P (December 14, 1998)

Search Selected Search ALL Clear

PATENT-FAMILY:

	PUB-NO	PUB-DATE	LANGUAGE	PAGES	MAIN-IPC
☐	JP 2002533063 W	October 8, 2002		125	C12N015/09
☐	WO 200035946 A1	June 22, 2000	E	127	C07K014/435
☐	EP 1141003 A1	October 10, 2001	E	000	C07K014/435

INT-CL (IPC): A61 K 45/00; A61 P 3/10; A61 P 9/10; A61 P 35/00; A61 P 43/00; C07 K 14/435; C07 K 19/00; C12 N 1/15; C12 N 1/19; C12 N 1/21; C12 N 5/10; C12 N 9/12; C12 N 15/09; C12 N 15/54; C12 N 15/63; C12 P 21/02; C12 Q 1/48; G01 N 33/15; G01 N 33/50

ABSTRACTED-PUB-NO: WO 200035946A
BASIC-ABSTRACT:

NOVELTY - A method (M1) for activating serum and glucocorticoid-induced protein kinase (SGK), where SGK is phosphorylated, is new.

DETAILED DESCRIPTION - INDEPENDENT CLAIMS are also included for the following:

(1) a method (M2) for reducing the activity of phosphorylated SGK where SGK is dephosphorylated;

(2) a fusion polypeptide (P1) comprising human SGK, or a fragment or variant;

(3) a polypeptide (P2) comprising human SGK, where the residue equivalent to serine 422, threonine 256 or lysine 127 of the full length human SGK1 is replaced;

(4) a polynucleotide (N1 and N2) encoding P1 and P2;

(5) a recombinant polynucleotide suitable for expressing P1 or P2;

(6) a host cell comprising N1 or N2;

(7) a method (M3) of making P1 or P2 comprising culturing the host cell of (6);

(8) a polypeptide obtained by M3;

(9) a method (M4) of identifying a compound that modulates the activity of SGK, where activated SGK is used;

(10) a method (M5) of identifying a compound which binds to a physiological substrate of SGK, such as BAD or GSK3, and either enhances or prevents its activation and/or phosphorylation by SGK;

(11) a method (M6) of identifying a compound which modulates the activation of SGK by an interacting polypeptide, such as PDK1 or a polypeptide with PDK2 activity;

(12) a method (M7) of identifying a polypeptide that interacts with activated SGK;

(13) a kit for carrying out the methods of M4-M7; and

(14) compounds identified by the methods M4-M7.

ACTIVITY - Cytostatic; antidiabetic; vasotropic.

No biological data given

MECHANISM OF ACTION - Modulation of serum and glucocorticoid-induced protein kinase (SGK).

USE - The compounds identified by M4-M7 are useful for treating patients requiring modulation of SGK, such as patients with cancer, diabetes or ischemic disease. PP1, PP2C or PP2A are useful for deactivating and/or dephosphorylating SGK. PDK1 is useful for activating and/or phosphorylating SGK.

ABSTRACTED-PUB-NO: WO 200035946A
EQUIVALENT-ABSTRACTS:

CHOSEN-DRAWING: Dwg.0/20

Previous Doc Next Doc Go to Doc#

WEST Search History

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=> s (SGK-1 or SGK-2) and protein kinase and activation
L1          15 (SGK-1 OR SGK-2) AND PROTEIN KINASE AND ACTIVATION

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2             6 DUP REM L1 (9 DUPLICATES REMOVED)

=> d l2 1-6 ibib ab

L2   ANSWER 1 OF 6  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:         2004:824055  HCAPLUS
DOCUMENT NUMBER:          141:330185
TITLE:                    Gene expression profiling for diagnosis and treatment
                          of angiogenesis-related disorders
INVENTOR(S):              Gonda, Thomas John; Kremmidiotis, Gabriel
PATENT ASSIGNEE(S):       Bionomics Limited, Australia
SOURCE:                   PCT Int. Appl., 148 pp.
                          CODEN: PIXXD2
DOCUMENT TYPE:            Patent
LANGUAGE:                 English
FAMILY ACC. NUM. COUNT:   1
PATENT INFORMATION:

     PATENT NO.           KIND   DATE        APPLICATION NO.         DATE
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     WO 2004085675         A1    20041007    WO 2004-AU383           20040326
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             GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC,
             LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NA, NI,
             NO, NZ, OM, PG, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, SY,
             TJ, TM, TN, TR, TT, TZ, UA, UG, US, UZ, VC, VN, YU, ZA, ZM, ZW
         RW: BW, GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ,
             BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE,
             ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PL, PT, RO, SE, SI,
             SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN,
             TD, TG
PRIORITY APPLN. INFO.:                       AU 2003-901511      A  20030328
AB   The present invention provides methods of gene expression profiling for
     diagnosis and treatment of angiogenesis-related disorders.  Diseases of
     the invention include cancer, rhematoid arthritis, diabetic retinopathy,
```

psoriasis, cardiovascular diseases such as atherosclerosis, ischmeic limb disease and coronary heart disease.

REFERENCE COUNT: 4 THERE ARE 4 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 2 OF 6 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2004105412 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14996737
TITLE: Microarray analysis reveals glucocorticoid-regulated survival genes that are associated with inhibition of apoptosis in breast epithelial cells.
AUTHOR: Wu Wei; Chaudhuri Shamita; Brickley Deanna R; Pang Diana; Karrison Theodore; Conzen Suzanne D
CORPORATE SOURCE: Department of Medicine, University of Chicago, Chicago, Illinois 60637, USA.
CONTRACT NUMBER: CA89208 (NCI)
CA90459 (NCI)
SOURCE: Cancer research, (2004 Mar 1) 64 (5) 1757-64.
Journal code: 2984705R. ISSN: 0008-5472.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200403
ENTRY DATE: Entered STN: 20040304
Last Updated on STN: 20040401
Entered Medline: 20040331

AB **Activation** of the glucocorticoid receptor (GR) results in diverse physiological effects depending on cell type. For example, glucocorticoids (GC) cause apoptosis in lymphocytes but can rescue mammary epithelial cells from growth factor withdrawal-induced death. However, the molecular mechanisms of GR-mediated survival remain poorly understood. In this study, a large-scale oligonucleotide screen of GR-regulated genes was performed. Several of the genes that were found to be induced 30 min after GR **activation** encode proteins that function in cell survival signaling pathways. We also demonstrate that dexamethasone pretreatment of breast cancer cell lines inhibits chemotherapy-induced apoptosis in a GR-dependent manner and is associated with the transcriptional induction of at least two genes identified in our screen, serum and GC-inducible **protein kinase**-1 (**SGK** -1) and mitogen-activated **protein kinase** phosphatase-1 (MKP-1). Furthermore, GC treatment alone or GC treatment followed by chemotherapy increases both **SGK-1** and MKP-1 steady-state protein levels. In the absence of GC treatment, ectopic expression of **SGK-1** or MKP-1 inhibits chemotherapy-induced apoptosis, suggesting a possible role for these proteins in GR-mediated survival. Moreover, specific inhibition of **SGK-1** or MKP-1 induction by the introduction of **SGK-1**- or MKP-1-small interfering RNA reversed the anti-apoptotic effects of GC treatment. Taken together, these data suggest that GR **activation** in breast cancer cells regulates survival signaling through direct transactivation of genes that encode proteins that decrease susceptibility to apoptosis. Given the widespread clinical administration of dexamethasone before chemotherapy, understanding GR-induced survival mechanisms is essential for achieving optimal therapeutic responses.

L2 ANSWER 3 OF 6 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 2004160628 EMBASE
TITLE: C. elegans **SGK-1** is the critical component in the Akt/PKB kinase complex to control stress response and life span.
AUTHOR: Hertweck M.; Gobel C.; Baumeister R.
CORPORATE SOURCE: R. Baumeister, BioIII, Bioinformatics/Molecular Genetics,

University of Freiburg, D-79104 Freiburg, Germany.
baumeister@celegans.de
SOURCE: Developmental Cell, (2004) 6/4 (577-588).
Refs: 32
ISSN: 1534-5807 CODEN: DCEEBE
PUBLISHER IDENT.: S 1534-5807(04)00095-4
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 004 Microbiology
021 Developmental Biology and Teratology
LANGUAGE: English
SUMMARY LANGUAGE: English

AB The DAF-2 insulin receptor-like signaling pathway controls metabolism, development, longevity, and stress response in C. elegans . Here we show that **SGK-1**, the C. elegans homolog of the serum- and glucocorticoid-inducible kinase SGK, acts in parallel to the AKT kinases to mediate DAF-2 signaling. Loss of **sgk-1** results in defective egg-laying, extended generation time, increased stress resistance, and an extension of life span. **SGK-1** forms a protein complex with the AKT kinases, and is activated by and strictly depends on PDK-1. All three kinases of this complex are able to directly phosphorylate DAF-16/FKHRL1, yet have different functions in DAF-2 signaling. Whereas AKT-1 and AKT-2 are more important for regulating dauer formation, **SGK-1** is the crucial factor for the control of development, stress response, and longevity. Our data also suggest the existence of a second pathway from DAF-2 to DAF-16 that does not depend on AKT-1, AKT-2, and **SGK-1**. .COPYRGT. 2004 by Cell Press.

L2 ANSWER 4 OF 6 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2004:285436 BIOSIS
DOCUMENT NUMBER: PREV200400284193
TITLE: Transcriptional regulation of SGK survival kinase in human malignant cholangiocytes.
AUTHOR(S): Meng, Fanyin [Reprint Author]; Brooks, Linda; Chiasson, Valorie; Patel, Tushar C
CORPORATE SOURCE: Research and Education, Scott and White Memorial Hospital, 2401 South 31st Street, Temple, TX, 76508, USA
fmeng@swmail.sw.org
SOURCE: FASEB Journal, (2004) Vol. 18, No. 4-5, pp. Abst. 471.13.
http://www.fasebj.org/. e-file.
Meeting Info.: FASEB Meeting on Experimental Biology: Translating the Genome. Washington, District of Columbia, USA. April 17-21, 2004. FASEB.
ISSN: 0892-6638 (ISSN print).
DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LANGUAGE: English
ENTRY DATE: Entered STN: 16 Jun 2004
Last Updated on STN: 16 Jun 2004

AB The serum and glucocorticoid inducible **protein kinase** gene, SGK, has been implicated as a key component of the cellular survival response. We have shown that IL-6 stimulates survival signaling in malignant cholangiocytes. Furthermore, IL-6 activates p38 MAPK signaling in malignant but not in normal cholangiocytes. However, the regulation of SGK by IL-6 mediated p38 MAPK signaling is unknown. Our AIM was to study the mechanisms by which IL-6 and/or p38 MAP kinase regulate SGK activity. p38a MAPK was overexpressed using adenoviral constructs. **SGK-1** mRNA expression was quantitated by real-time PCR. Both IL-6 treatment and p38a overexpression increased **SGK-1** mRNA expression in KMCH-1 malignant human cholangiocytes. Furthermore dominant negative p38a down-regulated basal **SGK-1** protein and mRNA expression. Overexpression of p38a inhibited IL-6 stimulated **SGK-1** expression. Both the transcriptional inhibitor actinomycin D and the translational inhibitor cycloheximide successfully prevented the induction of **SGK-1** protein expression by

IL-6 and p38a overexpression. In contrast to its effect on **SGK-1**, neither IL-6 nor p38 MAPK altered expression of **SGK-2** and SGK-3 protein expression. Thus, **SGK-1** expression is regulated by IL-6 stimulation as well as by the p38a MAPK signaling. In conclusion, survival signaling by IL-6 in malignant cholangiocytes may involve transcriptional regulation of **SGK-1** via a p38 MAPK dependent pathway. (Supported by the Scott & White Hospital Foundation).

L2 ANSWER 5 OF 6 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2002654555 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12218062
TITLE: Ubiquitin modification of serum and glucocorticoid-induced **protein kinase**-1 (**SGK-1**).
AUTHOR: Brickley Deanna R; Mikosz Christina A; Hagan Christy R; Conzen Suzanne D
CORPORATE SOURCE: Department of Medicine, Section of Hematology/Oncology, University of Chicago, Chicago, Illinois 60637, USA.
CONTRACT NUMBER: CA14599-25 (NCI)
K08 CA90459 (NCI)
SOURCE: Journal of biological chemistry, (2002 Nov 8) 277 (45) 43064-70.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20021105
Last Updated on STN: 20030207
Entered Medline: 20030206

AB The serum and glucocorticoid-induced **protein kinase** gene (**sgk-1**) encodes a multifunctional kinase that can be phosphorylated and activated through a phosphatidylinositol 3-kinase-dependent signaling pathway. In many cell types, endogenous **SGK-1** steady-state protein levels are very low but can be acutely up-regulated after glucocorticoid receptor-mediated transcriptional **activation**; in breast epithelial and cancer cell lines, this up-regulation is associated with promotion of cell survival. We and others have noted that ectopically introduced full-length **SGK-1** is poorly expressed, although **SGK-1** lacking the first 60 amino acids (delta60SGK-1) is expressed at much higher-fold protein levels than wild-type **SGK-1** in both human embryonic kidney 293T and MCF10A mammary epithelial cells. In this report, we demonstrate for the first time that the low steady-state expression level of **SGK-1** is due to polyubiquitination and subsequent degradation by the 26S proteasome. Deletion of the amino-terminal 60 amino acids of **SGK-1** results in a mutant **SGK-1** protein that is neither efficiently polyubiquitinated nor degraded by the 26S proteasome, accounting for the higher steady-state levels of the truncated protein. We also demonstrate that a subset of **SGK-1** localizes to the plasma membrane and that the polyubiquitin-modified **SGK-1** localizes to a membrane-associated fraction of the cell. Taken together, these data suggest that a significant fraction of **SGK-1** is membrane-associated and ubiquitinated. These findings are consistent with the recently described role of **SGK-1** in phosphorylating the membrane-associated protein Nedd4-2 and the integral membrane Na+/H+ exchanger isoform 3 (NHE3) and suggest a novel mechanism of regulation of **SGK-1**.

L2 ANSWER 6 OF 6 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 3
ACCESSION NUMBER: 2001:395086 HCAPLUS
DOCUMENT NUMBER: 135:102719

TITLE: Glucocorticoid receptor-mediated protection from apoptosis is associated with induction of the serine/threonine survival kinase gene, **sgk-1**

AUTHOR(S): Mikosz, Christina A.; Brickley, Deanna R.; Sharkey, Melinda S.; Moran, Timothy W.; Conzen, Suzanne D.

CORPORATE SOURCE: Department of Medicine, Section of Hematology / Oncology, University of Chicago, Chicago, IL, 60637, USA

SOURCE: Journal of Biological Chemistry (2001), 276(20), 16649-16654
CODEN: JBCHA3; ISSN: 0021-9258

PUBLISHER: American Society for Biochemistry and Molecular Biology

DOCUMENT TYPE: Journal

LANGUAGE: English

AB The authors previously demonstrated that **activation** of the glucocorticoid receptor (GR) initiates an anti-apoptotic signal in the immortalized human mammary epithelial cell line MCF10A that is dependent on the GR's transcriptional activity. In this study, the authors show that the survival role of GR **activation** extends to protecting human breast cancer cells undergoing apoptosis after growth factor deprivation. Serum and glucocorticoid-regulated kinase-1 (sgk), a gene previously identified as a direct transcriptional target of the activated GR in a rat mammary tumor cell line, was rapidly induced after GR **activation** in human mammary epithelial cells. Furthermore, in the absence of all growth factors, ectopic sgk expression inhibited apoptosis, suggesting that SGK is a survival kinase. Finally, kinase-dead SGK expression inhibited the protection from apoptosis usually seen after GR **activation**. These findings suggest that SGK is an important downstream target of GR-mediated survival signaling and that it is distinct from other survival kinases because it can be primarily regulated at the level of transcription.

REFERENCE COUNT: 21 THERE ARE 21 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

=> s (serum and glucocorticoid-induced protein kinase gene or sgk-1)
L3 89 (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR SGK-1)

=> s (serum and glucocorticoid-induced protein kinase gene or human sgk-1)
L4 8 (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR HUMAN SGK-1)

=> dup rem l4
PROCESSING COMPLETED FOR L4
L5 4 DUP REM L4 (4 DUPLICATES REMOVED)

=> d l5 1-4 ibib ab

L5 ANSWER 1 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2004:171531 BIOSIS
DOCUMENT NUMBER: PREV200400159126
TITLE: Microarray analysis reveals glucocorticoid-regulated survival genes that are associated with inhibition of apoptosis in breast epithelial cells.
AUTHOR(S): Wu, Wei; Chaudhuri, Shamita; Brickley, Deanna R.; Pang, Diana; Karrison, Theodore; Conzen, Suzanne D. [Reprint Author]
CORPORATE SOURCE: Department of Medicine, University of Chicago, 5841 S. Maryland Avenue, MC 2115, Chicago, IL, 60637, USA
sconzen@medicine.bsd.uchicago.edu
SOURCE: Cancer Research, (March 1 2004) Vol. 64, No. 5, pp. 1757-1764. print.
ISSN: 0008-5472 (ISSN print).

DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 24 Mar 2004
Last Updated on STN: 24 Mar 2004

AB Activation of the glucocorticoid receptor (GR) results in diverse physiological effects depending on cell type. For example, glucocorticoids (GC) cause apoptosis in lymphocytes but can rescue mammary epithelial cells from growth factor withdrawal-induced death. However, the molecular mechanisms of GR-mediated survival remain poorly understood. In this study, a large-scale oligonucleotide screen of GR-regulated genes was performed. Several of the genes that were found to be induced 30 min after GR activation encode proteins that function in cell survival signaling pathways. We also demonstrate that dexamethasone pretreatment of breast cancer cell lines inhibits chemotherapy-induced apoptosis in a GR-dependent manner and is associated with the transcriptional induction of at least two genes identified in our screen, serum and GC-inducible protein kinase-1 (SGK-1) and mitogen-activated protein kinase phosphatase-1 (MKP-1). Furthermore, GC treatment alone or GC treatment followed by chemotherapy increases both SGK-1 and MKP-1 steady-state protein levels. In the absence of GC treatment, ectopic expression of SGK-1 or MKP-1 inhibits chemotherapy-induced apoptosis, suggesting a possible role for these proteins in GR-mediated survival. Moreover, specific inhibition of SGK-1 or MKP-1 induction by the introduction of SGK-1- or MKP-1-small interfering RNA reversed the antiapoptotic effects of GC treatment. Taken together, these data suggest that GR activation in breast cancer cells regulates survival signaling through direct transactivation of genes that encode proteins that decrease susceptibility to apoptosis. Given the widespread clinical administration of dexamethasone before chemotherapy, understanding GR-induced survival mechanisms is essential for achieving optimal therapeutic responses.

L5 ANSWER 2 OF 4 MEDLINE on STN DUPLICATE 1
ACCESSION NUMBER: 2002654555 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12218062
TITLE: Ubiquitin modification of **serum** and glucocorticoid-induced protein kinase-1 (SGK-1).
AUTHOR: Brickley Deanna R; Mikosz Christina A; Hagan Christy R; Conzen Suzanne D
CORPORATE SOURCE: Department of Medicine, Section of Hematology/Oncology, University of Chicago, Chicago, Illinois 60637, USA.
CONTRACT NUMBER: CA14599-25 (NCI)
K08 CA90459 (NCI)
SOURCE: Journal of biological chemistry, (2002 Nov 8) 277 (45) 43064-70.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20021105
Last Updated on STN: 20030207
Entered Medline: 20030206

AB The **serum** and **glucocorticoid-induced protein kinase gene** (sgk-1) encodes a multifunctional kinase that can be phosphorylated and activated through a phosphatidylinositol 3-kinase-dependent signaling pathway. In many cell types, endogenous SGK-1 steady-state protein levels are very low but can be acutely up-regulated after glucocorticoid receptor-mediated transcriptional activation; in breast epithelial and cancer cell lines, this up-regulation is associated with promotion of cell survival. We and others have noted that ectopically introduced full-length SGK-1 is poorly expressed, although SGK-1 lacking the first 60 amino acids (delta60SGK-1) is expressed at much higher-fold protein levels than wild-type SGK-1 in both human embryonic kidney 293T and MCF10A mammary epithelial cells. In

this report, we demonstrate for the first time that the low steady-state expression level of SGK-1 is due to polyubiquitination and subsequent degradation by the 26S proteasome. Deletion of the amino-terminal 60 amino acids of SGK-1 results in a mutant SGK-1 protein that is neither efficiently polyubiquitinated nor degraded by the 26S proteasome, accounting for the higher steady-state levels of the truncated protein. We also demonstrate that a subset of SGK-1 localizes to the plasma membrane and that the polyubiquitin-modified SGK-1 localizes to a membrane-associated fraction of the cell. Taken together, these data suggest that a significant fraction of SGK-1 is membrane-associated and ubiquitinated. These findings are consistent with the recently described role of SGK-1 in phosphorylating the membrane-associated protein Nedd4-2 and the integral membrane Na+/H+ exchanger isoform 3 (NHE3) and suggest a novel mechanism of regulation of SGK-1.

L5 ANSWER 3 OF 4 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:783486 HCAPLUS
DOCUMENT NUMBER: 136:68047
TITLE: Alteration of cardiac and renal functions in transgenic mice overexpressing human mineralocorticoid receptor
AUTHOR(S): Le Menuet, Damien; Isnard, Richard; Bichara, Maurice; Viengchareun, Say; Muffat-Joly, Martine; Walker, Francine; Zennaro, Maria-Christina; Lombes, Marc
CORPORATE SOURCE: Faculte de Medecine Xavier Bichat, INSERM U478, Faculte de Medecine Xavier Bichat, Paris, 75018, Fr.
SOURCE: Journal of Biological Chemistry (2001), 276(42), 38911-38920
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The mineralocorticoid receptor (MR), a ligand-dependent transcription factor, mediates aldosterone actions in a large variety of tissues. To explore the functional implication of MR in pathophysiol., transgenic mouse models were generated using the proximal human MR (hMR) promoter to drive expression of hMR in aldosterone target tissues. Tissue-specific anal. of transgene expression in two independent transgenic animal (TG) lines by RNase protection assays revealed that hMR is expressed in all mineralocorticoid-sensitive tissues, most notably in the kidney and the heart. TG exhibit both renal and cardiac abnormalities. Enlarged kidneys were histol. assocd. with renal tubular dilation and cellular vacuolization whose prevalence increased with aging. Renal clearance studies also disclosed a significant decrease in urinary potassium excretion rate in TG. HMR-expressing animals had normal blood pressure but developed mild dilated cardiomyopathy (increased left ventricle diams. and decreased shortening fraction), which was accompanied by a significant increase in heart rate. Differential gene expression anal. revealed a 2- to 5-fold increase in cardiac expression of atrial natriuretic peptide, **serum**- and glucocorticoid-induced kinase, and early growth response gene 1 as detected by microarrays; renal **serum**- and glucocorticoid-induced kinase was also induced significantly. Altogether, TG exhibited specific alteration of renal and cardiac functions, thus providing useful pathophysiol. models to gain new insights into the tissue-specific mineralocorticoid signaling pathways.
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L5 ANSWER 4 OF 4 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2001:384797 BIOSIS
DOCUMENT NUMBER: PREV200100384797
TITLE: Glucocorticoid receptor-mediated protection from apoptosis is associated with induction of the serine/threonine survival kinase gene, sgk-1.

AUTHOR(S): Mikosz, Christina A.; Brickley, Deanna R.; Sharkey, Melinda S.; Moran, Timothy W.; Conzen, Suzanne D. [Reprint author]
CORPORATE SOURCE: Dept. of Medicine, University of Chicago, 5841 South Maryland Ave., Chicago, IL, 60637, USA
sconzen@medicine.bsd.uchicago.edu
SOURCE: Journal of Biological Chemistry, (May 18, 2001) Vol. 276, No. 20, pp. 16649-16654. print.
CODEN: JBCHA3. ISSN: 0021-9258.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 15 Aug 2001
Last Updated on STN: 19 Feb 2002

AB We previously demonstrated that activation of the glucocorticoid receptor (GR) initiates an antiapoptotic signal in the immortalized human mammary epithelial cell line MCF10A that is dependent on the GR's transcriptional activity. In this study, we show that the survival role of GR activation extends to protecting human breast cancer cells undergoing apoptosis after growth factor deprivation. Serum and glucocorticoid-regulated kinase-1 (sgk), a gene previously identified as a direct transcriptional target of the activated GR in a rat mammary tumor cell line, was rapidly induced after GR activation in human mammary epithelial cells. Furthermore, in the absence of all growth factors, ectopic sgk expression inhibited apoptosis, suggesting that SGK is a survival kinase. Finally, kinase-dead SGK expression inhibited the protection from apoptosis usually seen after GR activation. These findings suggest that SGK is an important downstream target of GR-mediated survival signaling and that it is distinct from other survival kinases because it can be primarily regulated at the level of transcription.

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=> d his

     (FILE 'HOME' ENTERED AT 15:45:23 ON 14 FEB 2005)

     FILE 'MEDLINE, HCAPLUS, SCISEARCH, EMBASE, BIOSIS, BIOTECHDS' ENTERED AT
     15:47:11 ON 14 FEB 2005
L1            15 S (SGK-1 OR SGK-2) AND PROTEIN KINASE AND ACTIVATION
L2             6 DUP REM L1 (9 DUPLICATES REMOVED)
L3            89 S (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR SGK-
L4             8 S (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR HUMA
L5             4 DUP REM L4 (4 DUPLICATES REMOVED)

=> dup rem l3
PROCESSING COMPLETED FOR L3
L6            47 DUP REM L3 (42 DUPLICATES REMOVED)

=> s l6 and 1990-1998/py
   5 FILES SEARCHED...
L7            11 L6 AND 1990-1998/PY

=> d l7 1-11 ibib ab

L7   ANSWER 1 OF 11  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:         2000:151982  HCAPLUS
TITLE:                    Cloning of sgk serine-threonine protein kinase from
                          shark rectal gland - a gene induced by hypertonicity
                          and secretagogues
AUTHOR(S):                Waldegger, Siegfried; Barth, Petra; Forrest, John N.,
                          Jr.; Greger, Rainer; Lang, Florian
CORPORATE SOURCE:         Department of Physiology 1, University of Tubingen,
                          Tubingen, D-72076, Germany
SOURCE:                   Pfluegers Archiv (1998), 436(4), 575-580
                          CODEN: PFLABK; ISSN: 0031-6768
PUBLISHER:                Springer-Verlag
DOCUMENT TYPE:            Journal
```

LANGUAGE: English

AB Recently, the cell-vol.-regulated serine-threonine protein kinase h-sgk was cloned from a human hepatoma cell line. The sgk gene was shown to be induced by cell shrinkage in many different mammalian cell lines. In this study, two highly conserved serine-threonine protein kinases, **sgk -1** and sgk-2, were cloned from rectal gland tissue of the spiny dogfish (Squalus acanthias). Both kinases showed a distinct pattern of tissue specificity, with high expression levels in kidney, intestine, liver and heart. In rectal gland slices **sgk-1** transcription was induced by exposure to hypertonic soln., redn. of the extracellular urea concn., and addn. of the secretagogues vasoactive intestinal polypeptide (VIP) and carbachol. The shark **sgk-1** serine-threonine protein kinase may therefore provide a link between cell vol., Cl-secretion and protein phosphorylation state in shark rectal gland cells.

REFERENCE COUNT: 35 THERE ARE 35 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L7 ANSWER 2 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1998:500176 HCAPLUS

DOCUMENT NUMBER: 129:217946

TITLE: Modeling of heat and mass transfer in intumescent fire-resistant coatings

AUTHOR(S): Zverev, V. G.; Gol'din, V. D.; Nesmelov, V. V.; Tsimbalyuk, A. F.

CORPORATE SOURCE: Tomsk. Gos. Univ., Tomsk, 634050, Russia

SOURCE: Fizika Goreniya i Vzryva (**1998**), 34(2), 90-98
CODEN: FGVZA7; ISSN: 0430-6228

PUBLISHER: Izdatel'stvo Sibirskogo Otdeleniya RAN

DOCUMENT TYPE: Journal

LANGUAGE: Russian

AB Heat transfer, mass transfer, and properties of a series of intumescent coatings was studied. Exptl. data are given on the wt. loss and degree of swelling of these materials as a function of temp. The mechanisms of fireproofing effect of intumescent coatings is analyzed. A math. model is presented, which makes it possible to prognosticate the state of fireproofed constructions after being subjected to heat stress characteristic for fires. The theor. calcns. were in good agreement with exptl. results obtained for steel sample.

L7 ANSWER 3 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:681025 HCAPLUS

DOCUMENT NUMBER: 127:348581

TITLE: Conversion of gases from petroleum processing to gasoline component hydrocarbons on a modified zeolite-containing catalyst

AUTHOR(S): Vaabel, A. S.; Dubenkova, L. B.; Deikina, V. S.; Romanenko, L. S.; Tselyutina, M. I.; Kutuzov, V. M.; Latyshev, V. P.

CORPORATE SOURCE: Inst. Nefte- Uglekhim. Sint., Irkutsk. Gos. Univ., Irkutsk, Russia

SOURCE: Neftekhimiya (**1997**), 37(3), 208-215
CODEN: NEFTAH; ISSN: 0028-2421

PUBLISHER: Nauka

DOCUMENT TYPE: Journal

LANGUAGE: Russian

AB Addns. of P 1.5, Zn 3, and La 1 wt.% to 5 wt.% Cr2O3-**SGK 1** zeolite catalyst increases the selectivity and yield of refinery gas (contg. 60-60% olefins) conversion to gasoline-range hydrocarbons at 300-550.degree. and 500-1000 h-1. The product has an octane no. of 80-82.

L7 ANSWER 4 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1997:319485 HCAPLUS

DOCUMENT NUMBER: 126:318663

TITLE: Hydrogen effect on C2-C4 hydrocarbon gas transformation oligomerization
AUTHOR(S): Vaabel, A. S.; Kutuzov, V. S.; Deikina, V. S.; Dubenkova, L. B.; Romanenko, L. S.; Tselyutina, M. I.; Latyshev, V. P.
CORPORATE SOURCE: Inst. Nefte-i Uglrkhim. Sint., Angarsk, Russia
SOURCE: Neftekhimiya (**1997**), 37(1), 49-55
CODEN: NEFTAH; ISSN: 0028-2421
PUBLISHER: Nauka
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The presence of hydrogen pos. affects C2-4 gaseous hydrocarbon transformation to liq. C5-10 hydrocarbons on Cr-modified zeolite catalysts, **SGK 1**, at 350.degree. increasing the of catalyst use prior to regeneration.

L7 ANSWER 5 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:401168 HCAPLUS
DOCUMENT NUMBER: 125:146605
TITLE: Effect of type of catalyst on the yield and characteristics of products of hydrocracking of naphtha fractions
AUTHOR(S): Prokopyuk, A. S.; Khavkin, V. A.; Aliev, R. R.; Nel'kenbaum, S. Ya.; Usmanov, R. M.
CORPORATE SOURCE: AO "Ufimskii NPZ", Ufa, Russia
SOURCE: Khimiya i Tekhnologiya Topliv i Masel (**1996**), (2), 18-20
CODEN: KTPMAG; ISSN: 0023-1169
PUBLISHER: Izdatel'stvo "Neft i Gaz"
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The hydrocracking of 3 naphtha fractions (b. 85-180, 105-180, and 120-180.degree.) was carried out in the presence of 5 zeolite-type catalysts based on high-silica (RZE-U-type) and ultrahigh silica (TsVM) zeolites and contg. different amts. and combinations of NiO, MoO3, and B2O5. The yield and characteristics (group compn., octane no., S content, etc.) of the products were detd. Based on catalyst evaluation the CKB-3M catalyst (contg. NiO, MoO3, and RZE-U-type high-silica zeolite) and the **SGK-1** catalyst (contg. MoO3 and TsVM-type ultrahigh-silica zeolite) were optimal.

L7 ANSWER 6 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 1996:80041 HCAPLUS
DOCUMENT NUMBER: 124:205526
TITLE: Method for hydrocarbon (HC) utilization from burning gases of oil-refining and other chemical industries
AUTHOR(S): Kutuzov, V. M.; Deikina, V. S.; Dubenkova, L. B.; Tselutina, M. I.; Romanenko, L. S.; Vaabel, A. S.; Latyshev, V. P.
CORPORATE SOURCE: Institute Coal and Oil-Chemical Synthesis, University Irkutsk (ICOS), Angarsk, 665813, Russia
SOURCE: Chemistry, Ecology, Health, Proceedings of the International Meeting [on] Zeolite Catalysis for the Solution of Environmental Problems, Yaroslavl, Russia, Jan. 6-12, 1992 (**1995**), Meeting Date 1992, 199-205. Editor(s): Ione, Kazimira Gavrilovna. Nova Science Publishers: Commack, N. Y.
CODEN: 62GZAP
DOCUMENT TYPE: Conference
LANGUAGE: English
AB C2-4 hydrocarbons were converted to C5-10 aliph. hydrocarbons or C6-12 arom. hydrocarbons in a flow-type reactor with stationary catalyst layer using Cr-modified zeolite catalyst.

L7 ANSWER 7 OF 11 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 1992:493384 HCAPLUS
DOCUMENT NUMBER: 117:93384
TITLE: New application of catalyst **SGK-1**
AUTHOR(S): Moreva, N. P.; Olenina, Z. K.; Yas'yan, Yu. P.; Adzhiev, A. Yu.
CORPORATE SOURCE: VNIPI Gaznepererabotka, USSR
SOURCE: Khimiya i Tekhnologiya Topliv i Masel (**1992**), (3), 26-7
CODEN: KTPMAG; ISSN: 0023-1169
DOCUMENT TYPE: Journal
LANGUAGE: Russian
AB The title catalyst (contg. pentasil-type zeolite in H form, Mo oxide 5-8%; formed with Al2O3, for selective hydrocracking is also highly active in decompn. (>95%) of Et mercaptan and COS at 200-250.degree.. The catalyst is thus recommended for use in purifn. of gas from org. S compds.

L7 ANSWER 8 OF 11 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1999:105384 SCISEARCH
THE GENUINE ARTICLE: 161CY
TITLE: Modeling heat and mass transfer in intumescent fire-retardant coatings
AUTHOR: Zverev V G (Reprint); Goldin V D; Nesmelov V V; Tsimbalyuk A F
CORPORATE SOURCE: TOMSK VV KUIBYSHEV STATE UNIV, INST APPL MATH & MECH, TOMSK 634050, RUSSIA (Reprint)
COUNTRY OF AUTHOR: RUSSIA
SOURCE: COMBUSTION EXPLOSION AND SHOCK WAVES, (**MAR-APR 1998**) Vol. 34, No. 2, pp. 198-205.
Publisher: PLENUM PUBL CORP, CONSULTANTS BUREAU, 233 SPRING ST, NEW YORK, NY 10013.
ISSN: 0010-5082.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: ENGI
LANGUAGE: English
REFERENCE COUNT: 11
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The heat- and mass-transfer processes and properties of the intumescent fire-retardant compositions OVR-1, 336-11-88, and **SGK-1** are studied. The mass loss and expansion ratio of the materials are determined experimentally as functions of temperature. Mechanisms of the fire-retardant effect of these materials are analyzed. A new mathematical model is proposed that permits predicting the state of protected structures under thermal loads typical of fire conditions, A comparison shows good agreement between the numerical-calculation results and data of fire tests.

L7 ANSWER 9 OF 11 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 97:787584 SCISEARCH
THE GENUINE ARTICLE: YB829
TITLE: Conversion of oil-refining gases into liquid hydrocarbon components of gasoline on a modified zeolite-containing catalyst
AUTHOR: Vaabel A S (Reprint); Dubenkova L B; Deikina V S; Romanenko L S; Tselyutina M I; Kutuzov V M; Latyshev V P
CORPORATE SOURCE: IRKUTSK STATE UNIV, INST PETRO & CARBON CHEM SYNTH, IRKUTSK 664003, RUSSIA (Reprint)
COUNTRY OF AUTHOR: RUSSIA
SOURCE: PETROLEUM CHEMISTRY, (**OCT 1997**) Vol. 37, No. 3, pp. 201-209.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB.
ISSN: 0965-5441.
DOCUMENT TYPE: Article; Journal

FILE SEGMENT: ENGI
LANGUAGE: English
REFERENCE COUNT: 8

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A study has been made of the effect of additions of phosphorus, lanthanum and zinc on the activity of zeolite-containing catalyst **SGK-1** modified with chromium in the conversion of a mixture of C-2-C-4 gases into liquid C-5-C-10 hydrocarbons (gasoline components). It has been established that the introduction of additives (P, La and Zn) increases the selectivity and yield of liquid hydrocarbons. The dependence of the composition of products of conversion of C-2-C-4 gases on the temperature and the time of operation of the catalysts has been studied.

L7 ANSWER 10 OF 11 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 97:497697 SCISEARCH
THE GENUINE ARTICLE: XG672
TITLE: Influence of hydrogen on the conversion of C-2-C-4 hydrocarbon gases during oligomerization
AUTHOR: Vaabel A S (Reprint); Kutuzov V M; Deikina V S; Dubenkova L B; Romanenko L S; Tselyutina M I; Latyshev V P
CORPORATE SOURCE: IRKUTSK STATE UNIV, INST PETR & CARBOCHEM SYNTH, ANGARSK, RUSSIA (Reprint)
COUNTRY OF AUTHOR: RUSSIA
SOURCE: PETROLEUM CHEMISTRY, (**MAY 1997**) Vol. 37, No. 1, pp. 51-58.
Publisher: PERGAMON-ELSEVIER SCIENCE LTD, THE BOULEVARD, LANGFORD LANE, KIDLINGTON, OXFORD, ENGLAND OX5 1GB.
ISSN: 0965-5441.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: ENGI
LANGUAGE: English
REFERENCE COUNT: 3

ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A study has been made of the influence of hydrogen on the conversion of a mixture of C-2-C-4 hydrocarbon gases into liquid C-5-C-12 hydrocarbons on an **SGK-1** zeolite-containing catalyst modified with chromium under oligomerization conditions (t = 350 degrees C). It has been established that the presence of hydrogen in C-2-C-4 gases has a positive effect on the production of C-5-C-10 aliphatic hydrocarbons and increases the period of active operation of a zeolite-containing catalyst between regenerations.

L7 ANSWER 11 OF 11 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 92:600368 SCISEARCH
THE GENUINE ARTICLE: JR775
TITLE: NEW TREND IN USING **SGK-1** CATALYSTS
AUTHOR: MOREVA N P (Reprint); OLENINA Z K; YASYAN Y P; ADZHIEV A Y
SOURCE: CHEMISTRY AND TECHNOLOGY OF FUELS AND OILS, (**MAR/APR 1992**) Vol. 28, No. 3-4, pp. 161-163.
ISSN: 0009-3092.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: ENGI
LANGUAGE: ENGLISH
REFERENCE COUNT: 5

=> s human sgk-1
L8 2 HUMAN SGK-1

=> s human sgk
L9 46 HUMAN SGK

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=> s l9 and 1990-1998/py
   5 FILES SEARCHED...
L10          7 L9 AND 1990-1998/PY

=> d l10 1-7 ibib ab

L10  ANSWER 1 OF 7     MEDLINE on STN
ACCESSION NUMBER:   1998390195     MEDLINE
DOCUMENT NUMBER:    PubMed ID: 9722955
TITLE:              Genomic organization and chromosomal localization of the
                    human SGK protein kinase gene.
AUTHOR:             Waldegger S; Erdel M; Nagl U O; Barth P; Raber G; Steuer S;
                    Utermann G; Paulmichl M; Lang F
CORPORATE SOURCE:   Department of Physiology I, University of Tubingen,
                    Germany.. siegfried.waldegger@uni-tuebingen.de
SOURCE:             Genomics, (1998 Jul 15) 51 (2) 299-302.
                    Journal code: 8800135. ISSN: 0888-7543.
PUB. COUNTRY:       United States
DOCUMENT TYPE:      Journal; Article; (JOURNAL ARTICLE)
LANGUAGE:           English
FILE SEGMENT:       Priority Journals
OTHER SOURCE:       GENBANK-AJ000512
ENTRY MONTH:        199811
ENTRY DATE:         Entered STN: 19990106
                    Last Updated on STN: 20020420
                    Entered Medline: 19981105
AB   The SGK protein kinase is a novel member of the serine/threonine protein
     kinase family. Its corresponding gene belongs to the group of
     immediate-early genes. SGK transcription is controlled by cell volume
     alterations in different cell lines. To analyze the genomic structure and
     chromosomal location of the SGK gene, a human P1 clone was isolated by
     screening a human genomic library with a SGK cDNA probe. This clone was
     confirmed to encode the authentic SGK gene by the detection of exon-intron
     structures and the correspondence between the nucleotide sequences of
     exons and human cDNA. Using this P1 clone as a probe for fluorescence in
     situ hybridization, a single chromosomal locus for SGK was assigned to
     band 6q23, a region frequently affected by deletion in various human
     neoplasms.

L10  ANSWER 2 OF 7  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:        1998:565048  HCAPLUS
DOCUMENT NUMBER:         129:271364
TITLE:                   Genomic organization and chromosomal localization of
                         the human SGK protein kinase gene
AUTHOR(S):               Waldegger, Siegried; Erdel, Martin; Nagl, Ulrich O.;
                         Barth, Petra; Raber, Gertraud; Steuer, Silvia;
                         Utermann, Gerd; Paulmichl, Markus; Lang, Florian
CORPORATE SOURCE:        Dep. Physiology I, Univ. Tubingen, Tubingen, D-72076,
                         Germany
SOURCE:                  Genomics (1998), 51(2), 299-302
                         CODEN: GNMCEP; ISSN: 0888-7543
PUBLISHER:               Academic Press
DOCUMENT TYPE:           Journal
LANGUAGE:                English
AB   The SGK protein kinase is a novel member of the serine/threonine protein
     kinase family. Its corresponding gene belongs to the group of
     immediate-early genes. SGK transcription is controlled by cell vol.
     alterations in different cell lines. To analyze the genomic structure and
     chromosomal location of the SGK gene, a human P1 clone was isolated by
     screening a human genomic library with a SGK cDNA probe. This clone was
     confirmed to encode the authentic SGK gene by the detection of exon-intron
     structures and the correspondence between the nucleotide sequences of
     exons and human cDNA. Using this P1 clone as a probe for fluorescence in
     situ hybridization, a single chromosomal locus for SGK was assigned to
     band 6q23, a region frequently affected by deletion in various human
```

neoplasms. (c) 1998 Academic Press.
REFERENCE COUNT: 17 THERE ARE 17 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L10 ANSWER 3 OF 7 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:643385 SCISEARCH
THE GENUINE ARTICLE: 111WK
TITLE: Genomic organization and chromosomal localization of the **human SGK** protein kinase gene
AUTHOR: Waldegger S (Reprint); Erdel M; Nagl U O; Barth P; Raber G; Steuer S; Utermann G; Paulmichl M; Lang F
CORPORATE SOURCE: UNIV TUBINGEN, INST PHYSIOL 1, DEPT PHYSIOL 1, GMELINSTR 5, D-72076 TUBINGEN, GERMANY (Reprint); INNSBRUCK UNIV, DEPT MED BIOL & HUMAN GENET, A-6020 INNSBRUCK, AUSTRIA; INNSBRUCK UNIV, DEPT PHYSIOL, A-6020 INNSBRUCK, AUSTRIA
COUNTRY OF AUTHOR: GERMANY; AUSTRIA
SOURCE: GENOMICS, (**15 JUL 1998**) Vol. 51, No. 2, pp. 299-302.
Publisher: ACADEMIC PRESS INC JNL-COMP SUBSCRIPTIONS, 525 B ST, STE 1900, SAN DIEGO, CA 92101-4495.
ISSN: 0888-7543.
DOCUMENT TYPE: Article; Journal
FILE SEGMENT: LIFE
LANGUAGE: English
REFERENCE COUNT: 17
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB The SGK protein kinase is a novel member of the serine/threonine protein kinase family. Its corresponding gene belongs to the group of immediate-early genes. SGK transcription is controlled by cell volume alterations in different cell lines. To analyze the genomic structure and chromosomal location of the SGK gene, a human P1 clone was isolated by screening a human genomic library with a SGK cDNA probe. This clone was confirmed to encode the authentic SGK gene by the detection of exon-intron structures and the correspondence between the nucleotide sequences of exons and human cDNA. Using this P1 clone as a probe for fluorescence in situ hybridization, a single chromosomal locus for SGK was assigned to band 6q23, a region frequently affected by deletion in various human neoplasms. (C) 1998 Academic Press.

L10 ANSWER 4 OF 7 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 97:761978 SCISEARCH
THE GENUINE ARTICLE: XY103
TITLE: **Human SGK** - a putative serine/threonine kinase regulated by cell volume.
AUTHOR: Waldegger S (Reprint); Raber G; Steuer S; Risler T; Barth P; Lang F
CORPORATE SOURCE: UNIV TUBINGEN, DEPT PHYSIOL & INTERNAL MED, D-72074 TUBINGEN, GERMANY
COUNTRY OF AUTHOR: GERMANY
SOURCE: JOURNAL OF THE AMERICAN SOCIETY OF NEPHROLOGY, (**SEP 1997**) Vol. 8, Supp. [S], pp. A0273-A0273.
Publisher: WILLIAMS & WILKINS, 351 WEST CAMDEN ST, BALTIMORE, MD 21201-2436.
ISSN: 1046-6673.
DOCUMENT TYPE: Conference; Journal
FILE SEGMENT: LIFE; CLIN
LANGUAGE: English
REFERENCE COUNT: 0

L10 ANSWER 5 OF 7 EMBASE COPYRIGHT 2005 ELSEVIER INC. ALL RIGHTS RESERVED. on STN
ACCESSION NUMBER: 1998289648 EMBASE
TITLE: Genomic organization and chromosomal localization of the

human SGK protein kinase gene.
AUTHOR: Waldegger S.; Erdel M.; Nagl U.O.; Barth P.; Raber G.; Steuer S.; Utermann G.; Paulmichl M.; Lang F.
CORPORATE SOURCE: S. Waldegger, Physiologisches Institut I, Universitat Tubingen, Gmelinstr. 5, D-72076 Tubingen, Germany. siegfried.waldegger@uni-tuebingen.de
SOURCE: Genomics, (15 Jul 1998) 51/2 (299-302).
Refs: 17
ISSN: 0888-7543 CODEN: GNMCEP
COUNTRY: United States
DOCUMENT TYPE: Journal; Article
FILE SEGMENT: 022 Human Genetics
LANGUAGE: English
SUMMARY LANGUAGE: English
AB The SGK protein kinase is a novel member of the serine/threonine protein kinase family. Its corresponding gene belongs to the group of immediate-early genes. SGK transcription is controlled by cell volume alterations in different cell lines. To analyze the genomic structure and chromosomal location of the SGK gene, a human P1 clone was isolated by screening a human genomic library with a SGK cDNA probe. This clone was confirmed to encode the authentic SGK gene by the detection of exon-intron structures and the correspondence between the nucleotide sequences of exons and human cDNA. Using this P1 clone as a probe for fluorescence in situ hybridization, a single chromosomal locus for SGK was assigned to band 6q23, a region frequently affected by deletion in various human neoplasms.

L10 ANSWER 6 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:404848 BIOSIS
DOCUMENT NUMBER: PREV199800404848
TITLE: Genomic organization and chromosomal localization of the **human SGK** protein kinase gene.
AUTHOR(S): Waldegger, Siegfried [Reprint author]; Erdel, Martin; Nagl, Ulrich O.; Barth, Petra; Raber, Gertraud; Steuer, Silvia; Utermann, Gerd; Paulmichl, Markus; Lang, Florian
CORPORATE SOURCE: Physiol. Inst. I, Univ. Tuebingen, Gmelinstr. 5, D-72076 Tuebingen, Germany
SOURCE: Genomics, (July 15, 1998) Vol. 51, No. 2, pp. 299-302. print.
CODEN: GNMCEP. ISSN: 0888-7543.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 21 Sep 1998
Last Updated on STN: 5 Nov 1998
AB The SGK protein kinase is a novel member of the serine/threonine protein kinase family. Its corresponding gene belongs to the group of immediate-early genes. SGK transcription is controlled by cell volume alterations in different cell lines. To analyze the genomic structure and chromosomal location of the SGK gene, a human P1 clone was isolated by screening a human genomic library with a SGK cDNA probe. This clone was confirmed to encode the authentic SGK gene by the detection of exon-intron structures and the correspondence between the nucleotide sequences of exons and human cDNA. Using this P1 clone as a probe for fluorescence in situ hybridization, a single chromosomal locus for SGK was assigned to band 6q23, a region frequently affected by deletion in various human neoplasms.

L10 ANSWER 7 OF 7 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 1998:22384 BIOSIS
DOCUMENT NUMBER: PREV199800022384
TITLE: **Human SGK**: A putative serine/threonine kinase regulated by cell volume.
AUTHOR(S): Waldegger, Siegfried; Raber, Gertraud; Steuer, Silvia; Risler, Teut; Barth, Petra; Lang, Florian
CORPORATE SOURCE: Dep. Physiol. and Intern. Med., Univ. Tueb., Tuebingen, Germany

SOURCE: Journal of the American Society of Nephrology, (Sept., 1997) Vol. 9, No. PROGRAM AND ABSTR. ISSUE, pp. 56A. print.
Meeting Info.: 30th Annual Meeting of the American Society of Nephrology. San Antonio, Texas, USA. November 2-5, 1997. American Society of Nephrology.
CODEN: JASNEU. ISSN: 1046-6673.

DOCUMENT TYPE: Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
Conference; (Meeting Poster)

LANGUAGE: English

ENTRY DATE: Entered STN: 5 Jan 1998
Last Updated on STN: 24 Feb 1998

=> d his

(FILE 'HOME' ENTERED AT 15:45:23 ON 14 FEB 2005)

FILE 'MEDLINE, HCAPLUS, SCISEARCH, EMBASE, BIOSIS, BIOTECHDS' ENTERED AT 15:47:11 ON 14 FEB 2005

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L1          15 S (SGK-1 OR SGK-2) AND PROTEIN KINASE AND ACTIVATION
L2           6 DUP REM L1 (9 DUPLICATES REMOVED)
L3          89 S (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR SGK-
L4           8 S (SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE GENE OR HUMA
L5           4 DUP REM L4 (4 DUPLICATES REMOVED)
L6          47 DUP REM L3 (42 DUPLICATES REMOVED)
L7          11 S L6 AND 1990-1998/PY
L8           2 S  HUMAN SGK-1
L9          46 S  HUMAN SGK
L10          7 S L9 AND 1990-1998/PY
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=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	113.12	113.75

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-8.03	-8.03

STN INTERNATIONAL LOGOFF AT 16:01:21 ON 14 FEB 2005

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=> file medline hcaplus biosis biotechds embase scisearch
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                         ENTRY    SESSION
FULL ESTIMATED COST                                       0.21       0.21

FILE 'MEDLINE' ENTERED AT 13:17:06 ON 14 FEB 2005

FILE 'HCAPLUS' ENTERED AT 13:17:06 ON 14 FEB 2005
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FILE 'SCISEARCH' ENTERED AT 13:17:06 ON 14 FEB 2005
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=> s serum and glucocorticoid-induced protein kinase
L1          62 SERUM AND GLUCOCORTICOID-INDUCED PROTEIN KINASE

=> dup rem l1
PROCESSING COMPLETED FOR L1
L2           22 DUP REM L1 (40 DUPLICATES REMOVED)

=> d l2 1-22 ibib ab

L2   ANSWER 1 OF 22  HCAPLUS  COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER:        2004:133687  HCAPLUS
DOCUMENT NUMBER:         141:274722
TITLE:                   Role of PDK1 in activating AGC protein kinase
AUTHOR(S):               Alessi, Dario R.
CORPORATE SOURCE:        MRC Protein Phosphorylation Unit, School of Life
                         Sciences, University of Dundee, Dundee, UK
SOURCE:                  Handbook of Cell Signaling (2004), Volume 1, 513-522.
                         Editor(s): Bradshaw, Ralph A.; Dennis, Edward A.
                         Elsevier: San Diego, Calif.
                         CODEN: 69FBUH; ISBN: 0-12-124546-2
DOCUMENT TYPE:           Conference; General Review
LANGUAGE:                English
AB   A review focuses on the research aimed at understanding the mechanism by
     which PtdIns (3,4,5)P3 (the phosphorylation product of phosphoinositide
     3-kinases and glycerophospholipid phosphatidylinositol 4,5-bisphosphate
     (PtdIns(4,5)P2) at the D-3 position of the inositol ring) regulates one
     branch of its downstream signaling pathways, namely enabling PDK1 to
     phosphorylate and activate a group of serine/threonine protein kinases
     that belong to the AGC subfamily of protein kinases.  These include
     isoforms of PKB, p70 ribosomal S6 kinase, serum- and
     glucocorticoid-induced protein kinase
     , p90 ribosomal S6 kinase, and protein kinase C isoforms.  Once these
     diverse AGC kinase members are activated, they phosphorylate and change
     the activity and function of key regulatory proteins that control
     processes such as cell proliferation and survival as well as cellular
     responses to insulin.
REFERENCE COUNT:         109   THERE ARE 109 CITED REFERENCES AVAILABLE FOR
                               THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE
                               FORMAT

L2   ANSWER 2 OF 22     MEDLINE on STN                    DUPLICATE 1
```

ACCESSION NUMBER: 2005024061 IN-PROCESS
DOCUMENT NUMBER: PubMed ID: 15650334
TITLE: 14-3-3 Protein mediates phosphorylation of microtubule-associated protein tau by **serum**- and **glucocorticoid-induced protein kinase** 1.
AUTHOR: Chun Jaesun; Kwon Taegun; Lee Eun Jeoung; Kim Chang Hyun; Han Yeon Soo; Hong Soon-Kwang; Hyun Sounghee; Kang Sang Sun
CORPORATE SOURCE: School of Science Education, Chungbuk National University, Chongju 361-763, Korea.
SOURCE: Molecules and cells, (2004 Dec 31) 18 (3) 360-8. Journal code: 9610936. ISSN: 1016-8478.
PUB. COUNTRY: Korea (South)
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20050115
Last Updated on STN: 20050129

AB The microtubule-associated protein, tau, is involved in numerous neuronal processes such as vesicle transport, microtubule-plasma membrane interaction and the intracellular localization of proteins. Tau is known to be phosphorylated by several kinases such as mitogen activated protein kinase, microtubule affinity regulating kinase, and protein kinase A. We found a putative **serum**- and **glucocorticoid-induced protein kinase** 1 (SGK1) phosphorylation site within the 207GSRSRTPSLP216 tau amino acid sequence. We report here that SGK1 phosphorylates Ser214 of Tau. Using a pull-down assay, we found that 14-3-3q interacts with SGK1 and tau to form a ternary protein complex that leads to phosphorylation of tau. 14-3-3 and phosphorylated tau were mainly co-localized in the nucleus of COS-1 cells. These results demonstrate that 14-3-3 scaffolds tau with SGK1 to facilitate the phosphorylation of tau at Ser214 and to regulate its subcellular localization.

L2 ANSWER 3 OF 22 MEDLINE on STN DUPLICATE 2
ACCESSION NUMBER: 2004060524 MEDLINE
DOCUMENT NUMBER: PubMed ID: 14611643
TITLE: WNK1, the kinase mutated in an inherited high-blood-pressure syndrome, is a novel PKB (protein kinase B)/Akt substrate.
AUTHOR: Vitari Alberto C; Deak Maria; Collins Barry J; Morrice Nick; Prescott Alan R; Phelan Anne; Humphreys Sian; Alessi Dario R
CORPORATE SOURCE: MRC Protein Phosphorylation Unit, School of Life Sciences, MSI/WTB complex, University of Dundee, Dow Street, Dundee DD1 5EH, Scotland, UK.. a.c.vitari@dundee.ac.uk
SOURCE: Biochemical journal, (2004 Feb 15) 378 (Pt 1) 257-68. Journal code: 2984726R. ISSN: 1470-8728.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200404
ENTRY DATE: Entered STN: 20040206
Last Updated on STN: 20040423
Entered Medline: 20040422

AB Recent evidence indicates that mutations in the gene encoding the WNK1 [with no K (lysine) protein kinase-1] results in an inherited hypertension syndrome called pseudohypoaldosteronism type II. The mechanisms by which WNK1 is regulated or the substrates it phosphorylates are currently unknown. We noticed that Thr-60 of WNK1, which lies N-terminal to the catalytic domain, is located within a PKB (protein kinase B) phosphorylation consensus sequence. We found that PKB phosphorylated WNK1 efficiently compared with known substrates, and both peptide map and mutational analysis revealed that the major PKB site of phosphorylation

was Thr-60. Employing a phosphospecific Thr-60 WNK1 antibody, we demonstrated that IGF1 (insulin-like growth factor) stimulation of HEK-293 cells induced phosphorylation of endogenously expressed WNK1 at Thr-60. Consistent with PKB mediating this phosphorylation, inhibitors of PI 3-kinase (phosphoinositide 3-kinase; wortmannin and LY294002) but not inhibitors of mammalian target of rapamycin (rapamycin) or MEK1 (mitogen-activated protein kinase kinase-1) activation (PD184352), inhibited IGF1-induced phosphorylation of endogenous WNK1 at Thr-60. Moreover, IGF1-induced phosphorylation of endogenous WNK1 did not occur in PDK1-/- ES (embryonic stem) cells, in which PKB is not activated. In contrast, IGF1 still induced normal phosphorylation of WNK1 in PDK1(L155E/L155E) knock-in ES cells in which PKB, but not S6K (p70 ribosomal S6 kinase) or SGK1 (**serum**- and **glucocorticoid** -**induced protein kinase** 1), is activated.
Our study provides strong pharmacological and genetic evidence that PKB mediates the phosphorylation of WNK1 at Thr-60 in vivo. We also performed experiments which suggest that the phosphorylation of WNK1 by PKB is not regulating its kinase activity or cellular localization directly. These results provide the first connection between the PI 3-kinase/PKB pathway and WNK1, suggesting a mechanism by which this pathway may influence blood pressure.

L2 ANSWER 4 OF 22 MEDLINE on STN DUPLICATE 3
ACCESSION NUMBER: 2004307358 MEDLINE
DOCUMENT NUMBER: PubMed ID: 15209375
TITLE: PDK1, the master regulator of AGC kinase signal transduction.
AUTHOR: Mora Alfonso; Komander David; van Aalten Daan M F; Alessi Dario R
CORPORATE SOURCE: MRC Protein Phosphorylation Unit, MSI/WTB Complex, University of Dundee, Dow Street, Dundee DD1 5EH, Scotland, UK.
SOURCE: Seminars in cell & developmental biology, (2004 Apr) 15 (2) 161-70. Ref: 74
Journal code: 9607332. ISSN: 1084-9521.
PUB. COUNTRY: England: United Kingdom
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200410
ENTRY DATE: Entered STN: 20040624
Last Updated on STN: 20041008
Entered Medline: 20041007

AB The interaction of insulin and growth factors with their receptors on the outside surface of a cell, leads to the activation of phosphatidylinositol 3-kinase (PI 3-kinase) and generation of the phosphatidylinositol 3,4,5-trisphosphate (PtdIns(3,4,5)P3) second messenger at the inner surface of the cell membrane. One of the most studied signalling events controlled by PtdIns(3,4,5)P3, comprises the activation of a group of AGC family protein kinases, including isoforms of protein kinase B (PKB)/Akt, p70 ribosomal S6 kinase (S6K), **serum**- and **glucocorticoid** -**induced protein kinase** (SGK) and protein kinase C (PKC), which play crucial roles in regulating physiological processes relevant to metabolism, growth, proliferation and survival. Here, we review recent biochemical, genetic and structural studies on the 3-phosphoinositide-dependent protein kinase-1 (PDK1), which phosphorylates and activates the AGC kinase members regulated by PI 3-kinase. We also discuss whether inhibitors of PDK1 might have chemotherapeutic potential in the treatment of cancers in which the PDK1-regulated AGC kinases are constitutively activated.

L2 ANSWER 5 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:950991 HCAPLUS

DOCUMENT NUMBER: 140:13034
TITLE: Methods for identifying roles of IKK.alpha., IKK.beta. and NEMO/IKK.gamma. kinases in inflammatory response and their use in screening for inflammatory diseases and cancer therapeutics
INVENTOR(S): Li, Jun; Marcu, Kenneth; Hanidu, Adedayo; Li, Xiang; Peet, Gregory; Mische, Sheenan
PATENT ASSIGNEE(S): Boehringer Ingelheim Pharmaceuticals, Inc., USA
SOURCE: PCT Int. Appl., 52 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
WO 2003099781	A2	20031204	WO 2003-US16586	20030523
WO 2003099781	A3	20040513		
W: AE, AG, AL, AM, AT, AU, AZ, BA, BB, BG, BR, BY, BZ, CA, CH, CN, CO, CR, CU, CZ, DE, DK, DM, DZ, EC, EE, ES, FI, GB, GD, GE, GH, GM, HR, HU, ID, IL, IN, IS, JP, KE, KG, KP, KR, KZ, LC, LK, LR, LS, LT, LU, LV, MA, MD, MG, MK, MN, MW, MX, MZ, NI, NO, NZ, OM, PH, PL, PT, RO, RU, SC, SD, SE, SG, SK, SL, TJ, TM, TN, TR, TT, TZ, UA, UG, UZ, VC, VN, YU, ZA, ZM, ZW				
RW: GH, GM, KE, LS, MW, MZ, SD, SL, SZ, TZ, UG, ZM, ZW, AM, AZ, BY, KG, KZ, MD, RU, TJ, TM, AT, BE, BG, CH, CY, CZ, DE, DK, EE, ES, FI, FR, GB, GR, HU, IE, IT, LU, MC, NL, PT, RO, SE, SI, SK, TR, BF, BJ, CF, CG, CI, CM, GA, GN, GQ, GW, ML, MR, NE, SN, TD, TG				
US 2004014111	A1	20040122	US 2003-446045	20030523
PRIORITY APPLN. INFO.:			US 2002-383018P P	20020524
			US 2002-406935P P	20020829

AB The invention provides methods for identifying roles of IKK.alpha., IKK.beta. and NEMO/IKK.gamma. kinases in inflammatory response and their use in screening for inflammatory diseases and cancer therapeutics. The method for identifying genes involved in the NF-.kappa.B pathway comprised of the steps of detg. the level of expression of a gene in an exptl. sample obtained from the cells having deficient levels of a component of the NF-.kappa.B pathway, detg. the level of expression of said gene in a control sample obtained from wild type cells having levels of a component of a biol. pathway, selecting genes having a level of expression that are modulated in said exptl. sample relative to said wild type sample.

L2 ANSWER 6 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:524617 HCAPLUS
DOCUMENT NUMBER: 139:194864
TITLE: Differing roles of Akt and **serum**- and glucocorticoid-regulated kinase in glucose metabolism, DNA synthesis, and oncogenic activity
AUTHOR(S): Sakoda, Hideyuki; Gotoh, Yukiko; Katagiri, Hideki; Kurokawa, Mineo; Ono, Hiraku; Onishi, Yukiko; Anai, Motonobu; Ogihara, Takehide; Fujishiro, Midori; Fukushima, Yasushi; Abe, Miho; Shojima, Nobuhiro; Kikuchi, Masatoshi; Oka, Yoshitomo; Hirai, Hisamaru; Asano, Tomoichiro
CORPORATE SOURCE: Institute for Adult Diseases, Asahi Life Foundation, 1-9-14 Nishishinjuku, Shinjuku-ku, Tokyo, 116, Japan
SOURCE: Journal of Biological Chemistry (2003), 278(28), 25802-25807
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB **Serum**- and glucocorticoid-regulated kinase (SGK) is a serine

kinase that has a catalytic domain homologous to that of Akt, but lacks the pleckstrin homol. domain present in Akt. Akt reportedly plays a key role in various cellular actions, including glucose transport, glycogen synthesis, DNA synthesis, anti-apoptotic activity, and cell proliferation. In this study, we attempted to reveal the different roles of SGK and Akt by overexpressing active mutants of Akt and SGK. We found that adenovirus-mediated overexpression of myristoylated (myr-) forms of Akt resulted in high glucose transport activity in 3T3-L1 adipocytes, phosphorylated glycogen synthase kinase-3 (GSK3) and enhanced glycogen synthase activity in hepatocytes, and the promotion of DNA synthesis in interleukin-3-dependent 32D cells. In addn., stable transfection of myr-Akt in NIH3T3 cells induced an oncogenic transformation in soft agar assays. The active mutant of SGK (D-SGK, substitution of Ser422 with Asp) and myr-SGK were shown to phosphorylate GSK3 and to enhance glycogen synthase activity in hepatocytes in a manner very similar to that obsd. for myr-Akt. However, despite the comparable degree of GSK3 phosphorylation between myr-Akt and D-SGK or myr-SGK, D-SGK and myr-SGK failed to enhance glucose transport activity in 3T3-L1 cells, DNA synthesis in 32D cells, and oncogenic transformation in NIH3T3 cells. Therefore, the different roles of SGK and Akt cannot be attributed to ability or inability to translocate to the membrane thorough the pleckstrin homol. domain, but rather must be attributable to differences in the relatively narrow substrate specificities of these kinases. In addn., our observations strongly suggest that phosphorylation of GSK3 is either not involved in or not sufficient for GLUT4 translocation, DNA synthesis, or oncogenic transformation. Thus, the identification of substrates selectively phosphorylated by Akt, but by not SGK, may provide clues to clarifying the pathway leading from Akt activation to these cellular activities.

REFERENCE COUNT: 49 THERE ARE 49 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 7 OF 22 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN

ACCESSION NUMBER: 2003:574891 BIOSIS
DOCUMENT NUMBER: PREV200300580137
TITLE: Insulin's expanding control of forkheads.
AUTHOR(S): Czech, Michael P. [Reprint Author]
CORPORATE SOURCE: Program in Molecular Medicine, University of Massachusetts Medical School, Worcester, MA, 01605, USA
michael.czech@umassmed.edu
SOURCE: Proceedings of the National Academy of Sciences of the United States of America, (September 30 2003) Vol. 100, No. 20, pp. 11198-11200. print.
ISSN: 0027-8424 (ISSN print).
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 10 Dec 2003
Last Updated on STN: 10 Dec 2003

L2 ANSWER 8 OF 22 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN DUPLICATE 4

ACCESSION NUMBER: 2003:512061 BIOSIS
DOCUMENT NUMBER: PREV200300515120
TITLE: In vivo role of the PIF-binding docking site of PDK1 defined by knock-in mutation.
AUTHOR(S): Collins, Barry J. [Reprint Author]; Deak, Maria; Arthur, J. Simon C.; Armit, Laura J.; Alessi, Dario R.
CORPORATE SOURCE: MRC Protein Phosphorylation Unit, University of Dundee, Dow Street, Dundee, DD1 5EH, UK
b.j.collins@dundee.ac.uk
SOURCE: EMBO (European Molecular Biology Organization) Journal, (August 15 2003) Vol. 22, No. 16, pp. 4202-4211. print.
ISSN: 0261-4189 (ISSN print).
DOCUMENT TYPE: Article

LANGUAGE: English
ENTRY DATE: Entered STN: 5 Nov 2003
Last Updated on STN: 5 Nov 2003

AB PKB/Akt, S6K, SGK and RSK are mediators of responses triggered by insulin and growth factors and are activated following phosphorylation by 3-phosphoinositide-dependent protein kinase-1 (PDK1). To investigate the importance of a substrate-docking site in the kinase domain of PDK1 termed the 'PIF-pocket', we generated embryonic stem (ES) cells in which both copies of the PDK1 gene were altered by knock-in mutation to express a form of PDK1 retaining catalytic activity, in which the PIF-pocket site was disrupted. The knock-in ES cells were viable, mutant PDK1 was expressed at normal levels and insulin-like growth factor 1 induced normal activation of PKB and phosphorylation of the PKB substrates GSK3 and FKHR. In contrast, S6K, RSK and SGK were not activated, nor were physiological substrates of S6K and RSK phosphorylated. These experiments establish the importance of the PIF-pocket in governing the activation of S6K, RSK, SGK, but not PKB, in vivo. They also illustrate the power of knock-in technology to probe the physiological roles of docking interactions in regulating the specificity of signal transduction pathways.

L2 ANSWER 9 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2003:277971 HCAPLUS
DOCUMENT NUMBER: 139:115133
TITLE: Importin-.alpha. mediates the regulated nuclear targeting of **serum**- and glucocorticoid-inducible protein kinase (Sgk) by recognition of a nuclear localization signal in the kinase central domain
AUTHOR(S): Maiyar, Anita C.; Leong, Meredith L. L.; Firestone, Gary L.
CORPORATE SOURCE: Department of Molecular and Cell Biology and The Cancer Research Laboratory, University of California at Berkeley, Berkeley, CA, 94720-3200, USA
SOURCE: Molecular Biology of the Cell (2003), 14(3), 1221-1239
CODEN: MBCEEV; ISSN: 1059-1524
PUBLISHER: American Society for Cell Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The transcriptionally regulated **serum** and glucocorticoid inducible protein kinase (Sgk) is localized to the nucleus in a **serum**-dependent manner, and a yeast two-hybrid genetic screen uncovered a specific interaction between Sgk and the importin-.alpha. nuclear import receptor. In vitro GST pull down assays demonstrated a strong and direct assocn. of importin-.alpha. with endogenous Sgk and exogenously expressed HA-tagged Sgk, whereas both components coimmunoppt. and colocalize to the nucleus after **serum** stimulation. Consistent with an active mechanism of nuclear localization, the nuclear import of HA-Sgk in permeabilized cells required ATP, cytoplasm, and a functional nuclear pore complex. Ectopic addn. of a 107 amino acid carboxy-terminal fragment of importin-.alpha., which contains the Sgk binding region, competitively inhibited the ability of endogenous importin-.alpha. to import Sgk into nuclei in vitro. Mutagenesis of lysines by alanine substitution defined a KKAILKKKEEK sequence within the central domain of Sgk between amino acids 131-141 that functions as a nuclear localization signal (NLS) required for the in vitro interaction with importin-.alpha. and for nuclear import of full-length Sgk in cultured cells. The **serum**-induced nuclear import of Sgk requires the NLS-dependent recognition of Sgk by importin-.alpha. as well as the PI3-kinase-dependent phosphorylation of Sgk. Our results define a new role importin-.alpha. in the stimulus-dependent control of signal transduction by nuclear localized protein kinases.

REFERENCE COUNT: 92 THERE ARE 92 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 10 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN

ACCESSION NUMBER: 2003:135245 HCAPLUS
DOCUMENT NUMBER: 138:363156
TITLE: Role of SGK in hormonal regulation of epithelial sodium channel in A6 cells
AUTHOR(S): De La Rosa, Diego Alvarez; Canessa, Cecilia M.
CORPORATE SOURCE: Department of Cellular and Molecular Physiology, Yale University School of Medicine, New Haven, CT, 06520-8026, USA
SOURCE: American Journal of Physiology (2003), 284(2, Pt. 1), C404-C414
CODEN: AJPHAP; ISSN: 0002-9513
PUBLISHER: American Physiological Society
DOCUMENT TYPE: Journal
LANGUAGE: English
AB The purpose of this study was to examine the role of the **serum**- and glucocorticoid-induced kinase (SGK) in the activation of the epithelial sodium channel (ENaC) by aldosterone, arginine vasopressin (AVP), and insulin. The authors used a tetracycline-inducible system to control the expression of wild-type (SGKwtT), constitutively active (S425D mutation; SGKS425DT), or inactive (K130M mutation; SGKK130MT) SGK in A6 cells independently of hormonal stimulation. The effect of SGK expression on ENaC activity was monitored by measuring transepithelial amiloride-sensitive short-circuit current (Isc) of transfected A6 cell lines. Expression of SGKwtT or SGKS425DT and aldosterone stimulation have additive effects on Isc. Although SGK could play some role in the aldosterone response, the authors' results suggest that other mechanisms take place. SGKS425DT abrogates the responses to AVP and insulin; hence, in the signaling pathways of these hormones there is a shared step that is stimulated by SGK. Because AVP and insulin induce fusion of vesicles to the apical membrane, the authors' results support the notion that SGK promotes incorporation of channels in the apical membrane.
REFERENCE COUNT: 33 THERE ARE 33 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 11 OF 22 MEDLINE on STN DUPLICATE 5
ACCESSION NUMBER: 2002654555 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12218062
TITLE: Ubiquitin modification of **serum** and **glucocorticoid-induced protein kinase**-1 (SGK-1).
AUTHOR: Brickley Deanna R; Mikosz Christina A; Hagan Christy R; Conzen Suzanne D
CORPORATE SOURCE: Department of Medicine, Section of Hematology/Oncology, University of Chicago, Chicago, Illinois 60637, USA.
CONTRACT NUMBER: CA14599-25 (NCI)
K08 CA90459 (NCI)
SOURCE: Journal of biological chemistry, (2002 Nov 8) 277 (45) 43064-70.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200302
ENTRY DATE: Entered STN: 20021105
Last Updated on STN: 20030207
Entered Medline: 20030206
AB The **serum** and **glucocorticoid-induced protein kinase** gene (sgk-1) encodes a multifunctional kinase that can be phosphorylated and activated through a phosphatidylinositol 3-kinase-dependent signaling pathway. In many cell types, endogenous SGK-1 steady-state protein levels are very low but can be acutely up-regulated after glucocorticoid receptor-mediated transcriptional activation; in breast epithelial and cancer cell lines, this up-regulation is associated with promotion of cell survival. We and

others have noted that ectopically introduced full-length SGK-1 is poorly expressed, although SGK-1 lacking the first 60 amino acids (delta60SGK-1) is expressed at much higher-fold protein levels than wild-type SGK-1 in both human embryonic kidney 293T and MCF10A mammary epithelial cells. In this report, we demonstrate for the first time that the low steady-state expression level of SGK-1 is due to polyubiquitination and subsequent degradation by the 26S proteasome. Deletion of the amino-terminal 60 amino acids of SGK-1 results in a mutant SGK-1 protein that is neither efficiently polyubiquitinated nor degraded by the 26S proteasome, accounting for the higher steady-state levels of the truncated protein. We also demonstrate that a subset of SGK-1 localizes to the plasma membrane and that the polyubiquitin-modified SGK-1 localizes to a membrane-associated fraction of the cell. Taken together, these data suggest that a significant fraction of SGK-1 is membrane-associated and ubiquitinated. These findings are consistent with the recently described role of SGK-1 in phosphorylating the membrane-associated protein Nedd4-2 and the integral membrane Na+/H+ exchanger isoform 3 (NHE3) and suggest a novel mechanism of regulation of SGK-1.

L2 ANSWER 12 OF 22 MEDLINE on STN DUPLICATE 6
ACCESSION NUMBER: 2002407794 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12023960
TITLE: Molecular basis for the substrate specificity of NIMA-related kinase-6 (NEK6). Evidence that NEK6 does not phosphorylate the hydrophobic motif of ribosomal S6 protein kinase and **serum**- and **glucocorticoid-induced protein kinase** in vivo.
AUTHOR: Lizcano Jose M; Deak Maria; Morrice Nick; Kieloch Agnieszka; Hastie C James; Dong Liying; Schutkowski Mike; Reimer Ulf; Alessi Dario R
CORPORATE SOURCE: Medical Research Council Protein Phosphorylation Unit, University of Dundee, Dow Street, Dundee DD1 5EH, Scotland, United Kingdom.. j.m.lizcano@dundee.ac.uk
SOURCE: Journal of biological chemistry, (2002 Aug 2) 277 (31) 27839-49.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200209
ENTRY DATE: Entered STN: 20020807
Last Updated on STN: 20030105
Entered Medline: 20020916

AB The AGC family of protein kinases, which includes isoforms of protein kinase B (also known as Akt), ribosomal S6 protein kinase (S6K), and **serum**- and **glucocorticoid-induced protein kinase** (SGK) are activated in response to many extracellular signals and play key roles in regulating diverse cellular processes. They are activated by the phosphorylation of the T loop of their kinase domain by the 3-phosphoinositide-dependent protein kinase-1 and by phosphorylation of a residue located C-terminal to the kinase domain in a region termed the hydrophobic motif. Recent work has implicated the NIMA (never in mitosis, gene A)-related kinase-6 (NEK6) as the enzyme that phosphorylates the hydrophobic motif of S6K1 in vivo. Here we demonstrate that in addition to phosphorylating S6K1 and SGK1 at their hydrophobic motif, NEK6 also phosphorylates S6K1 at two other sites and phosphorylates SGK1 at one other site in vitro. Employing the Jerini pepSTAR method in combination with kinetic analysis of phosphorylation of variant peptides, we establish the key substrate specificity determinants for NEK6. Our analysis indicates that NEK6 has a strong preference for Leu 3 residues N-terminal to the site of phosphorylation. Its mutation to either Ile or Val severely reduced the efficacy with which NEK6-phosphorylated peptide substrates, and moreover, mutation of the equivalent Leu residue in S6K1 or SGK1 prevented phosphorylation of their

hydrophobic motifs by NEK6 in vitro. However, these mutants of S6K1 or SGK1 still became phosphorylated at their hydrophobic motif following insulin-like growth factor-1 stimulation of transfected 293 cells. This study provides the first description of the basis for the substrate specificity of NEK6 and indicates that NEK6 is unlikely to be responsible for the IGF1-induced phosphorylation of the hydrophobic motif of S6K, SGK, and protein kinase B isoforms in vivo.

L2 ANSWER 13 OF 22 MEDLINE on STN DUPLICATE 7
ACCESSION NUMBER: 2002217165 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11842081
TITLE: The phosphatidylinositol 3-kinase (PI3K)-Akt pathway suppresses Bax translocation to mitochondria.
AUTHOR: Tsuruta Fuminori; Masuyama Norihisa; Gotoh Yukiko
CORPORATE SOURCE: Institute of Molecular and Cellular Biosciences, University of Tokyo, 1-1-1 Yayoi, Bunkyo-ku, Tokyo 113-0032, Japan.
SOURCE: Journal of biological chemistry, (2002 Apr 19) 277 (16) 14040-7.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200206
ENTRY DATE: Entered STN: 20020416
Last Updated on STN: 20030105
Entered Medline: 20020607

AB Bax, a proapoptotic member of the Bcl-2 family, localizes largely in the cytoplasm but redistributes to mitochondria in response to apoptotic stimuli, where it induces cytochrome c release. In this study, we show that the phosphatidylinositol 3-OH kinase (PI3K)-Akt pathway plays an important role in the regulation of Bax subcellular localization. We found that LY294002, a PI3K inhibitor, blocked the effects of **serum** to prevent Bax translocation to mitochondria and that expression of an active form of PI3K suppressed staurosporine-induced Bax translocation, suggesting that PI3K activity is essential for retaining Bax in the cytoplasm. In contrast, both U0126, a MEK inhibitor, and active MEK had little effect on Bax localization. In respect to downstream effectors of PI3K, we found that expression of active Akt, but not **serum** and **glucocorticoid-induced protein kinase** (SGK), suppressed staurosporine-induced translocation of Bax, whereas dominant negative Akt moderately promoted Bax translocation. Expression of Akt did not alter the levels of Bax, Bcl-2, Bcl-X(L), or phosphorylated JNK under the conditions used, suggesting that there were alternative mechanisms for Akt in the suppression of Bax translocation. Collectively, these results suggest that the PI3K-Akt pathway inhibits Bax translocation from cytoplasm to mitochondria and have revealed a novel mechanism by which the PI3K-Akt pathway promotes survival.

L2 ANSWER 14 OF 22 MEDLINE on STN DUPLICATE 8
ACCESSION NUMBER: 2002139112 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11751930
TITLE: Glucocorticoid activation of Na(+)/H(+) exchanger isoform 3 revisited. The roles of SGK1 and NHERF2.
AUTHOR: Yun C Chris; Chen Yueping; Lang Florian
CORPORATE SOURCE: Department of Medicine, Gastroenterology Division, The Johns Hopkins University School of Medicine, Baltimore, Maryland 21205, USA.. ccyun@emory.edu
CONTRACT NUMBER: DK-44484 (NIDDK)
SOURCE: Journal of biological chemistry, (2002 Mar 8) 277 (10) 7676-83.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)

LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200204
ENTRY DATE: Entered STN: 20020305
Last Updated on STN: 20030105
Entered Medline: 20020415

AB The stimulative effect of glucocorticoids on intestinal salt and water absorption has been known for more than two decades. However, molecular mechanisms underlying this activation remain elusive. Previous studies showed that methylprednisolone specifically increased Na(+)/H(+) exchanger isoform (NHE) 3 mRNA in ileum and kidney without affecting NHE1 mRNA levels. These results suggest that glucocorticoids activate NHE3 activity by induction of NHE3 transcripts. We recently found in PS120 and opossum kidney cells that chronic incubation with dexamethasone activated NHE3 independent of gene induction, indicating that the transcriptional activation may not be the only determining factor in the NHE3 activation. Furthermore, dexamethasone activated NHE3 activity only in the presence of a NHE3 regulatory protein, NHERF2, which was previously shown to confer cAMP-dependent inhibition of NHE3. This activation of NHE3 could not be duplicated by NHERF1. We identified **serum**- and **glucocorticoid-induced protein kinase**, SGK1, as the protein interacting with PDZ domains of NHERF2 to regulate NHE3 activity. The expression of SGK1 enhanced NHE3 transport in PS120 fibroblasts. In addition, the "kinase-dead" SGK1 blocked activation of NHE3 by dexamethasone in opossum kidney cells. These data demonstrated that glucocorticoid activation of NHE3 requires the activation of SGK1 and the presence of NHERF2 acting as a scaffold protein.

L2 ANSWER 15 OF 22 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
ACCESSION NUMBER: 2002:303625 BIOSIS
DOCUMENT NUMBER: PREV200200303625
TITLE: The insulin signalling pathway.
AUTHOR(S): Lizcano, Jose M. [Reprint author]; Alessi, Dario R. [Reprint author]
CORPORATE SOURCE: MRC Protein Phosphorylation Unit, School of Life Sciences, University of Dundee, MSI/WTB Complex, Dundee, DD1 5EH, UK d.r.alessi@dundee.ac.uk
SOURCE: Current Biology, (April 2, 2002) Vol. 12, No. 7, pp. R236-R238. print.
CODEN: CUBLE2. ISSN: 0960-9822.
DOCUMENT TYPE: Article
LANGUAGE: English
ENTRY DATE: Entered STN: 22 May 2002
Last Updated on STN: 22 May 2002

L2 ANSWER 16 OF 22 MEDLINE on STN DUPLICATE 9
ACCESSION NUMBER: 2002630189 MEDLINE
DOCUMENT NUMBER: PubMed ID: 12387817
TITLE: The Na(+)/H(+) exchanger regulatory factor 2 mediates phosphorylation of **serum**- and **glucocorticoid-induced protein kinase** 1 by 3-phosphoinositide-dependent protein kinase 1.
AUTHOR: Chun Jaesun; Kwon Taegun; Lee Eunjung; Suh Pann-Ghill; Choi Eui-Ju; Sun Kang Sang
CORPORATE SOURCE: School of Science Education, Chungbuk National University, Gaeshin-dong, Heungdok-gu, Chongju 361-763, Republic of Korea.
SOURCE: Biochemical and biophysical research communications, (2002 Oct 25) 298 (2) 207-15.
Journal code: 0372516. ISSN: 0006-291X.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 200211
ENTRY DATE: Entered STN: 20021022
Last Updated on STN: 20021214
Entered Medline: 20021126

AB The Na(+)/H(+) exchanger regulatory factor 2 (NHERF2/TKA-1/E3KARP) contains two PSD-95/Dlg/ZO-1 (PDZ) domains which interact with the PDZ docking motif (X-(S/T)-X-(V/L)) of proteins to mediate the assembly of transmembrane and cytosolic proteins into functional signal transduction complexes. One of the PDZ domains of NHERF2 interacts specifically with the DSLL, DSFL, and DTRL motifs present at the carboxy-termini of the 2-adrenergic receptor, the platelet-derived growth factor receptor, and the cystic fibrosis transmembrane conductance regulator, respectively. **Serum**- and **glucocorticoid-induced protein kinase** 1 (SGK1) also carries a putative PDZ-binding motif (D-S-F-L) at its carboxy tail, implicated in the specific interaction with NHERF2. There is a 3-phosphoinositide-dependent protein kinase 1 (PDK1) interacting fragment (PIF) in the tail of NHERF2. Using pull-down assays and co-transfection experiments, we demonstrated that the DSFL tail of SGK1 interacts with the first PDZ domain of NHERF2 and the PIF of NHERF2 binds to the PIF-binding pocket of PDK1 to form an SGK1-NHERF2-PDK1 complex. Formation of the protein complex promoted the phosphorylation and activation of SGK1 by PDK1. Thus, it was suggested that NHERF2 mediates the activation and phosphorylation of SGK1 by PDK1 through its first PDZ domain and PIF motif, as a novel SGK1 activation mechanism.

L2 ANSWER 17 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:783486 HCAPLUS
DOCUMENT NUMBER: 136:68047
TITLE: Alteration of cardiac and renal functions in transgenic mice overexpressing human mineralocorticoid receptor
AUTHOR(S): Le Menuet, Damien; Isnard, Richard; Bichara, Maurice; Viengchareun, Say; Muffat-Joly, Martine; Walker, Francine; Zennaro, Maria-Christina; Lombes, Marc
CORPORATE SOURCE: Faculte de Medecine Xavier Bichat, INSERM U478, Faculte de Medecine Xavier Bichat, Paris, 75018, Fr.
SOURCE: Journal of Biological Chemistry (2001), 276(42), 38911-38920
CODEN: JBCHA3; ISSN: 0021-9258
PUBLISHER: American Society for Biochemistry and Molecular Biology
DOCUMENT TYPE: Journal
LANGUAGE: English

AB The mineralocorticoid receptor (MR), a ligand-dependent transcription factor, mediates aldosterone actions in a large variety of tissues. To explore the functional implication of MR in pathophysiol., transgenic mouse models were generated using the proximal human MR (hMR) promoter to drive expression of hMR in aldosterone target tissues. Tissue-specific anal. of transgene expression in two independent transgenic animal (TG) lines by RNase protection assays revealed that hMR is expressed in all mineralocorticoid-sensitive tissues, most notably in the kidney and the heart. TG exhibit both renal and cardiac abnormalities. Enlarged kidneys were histol. assocd. with renal tubular dilation and cellular vacuolization whose prevalence increased with aging. Renal clearance studies also disclosed a significant decrease in urinary potassium excretion rate in TG. HMR-expressing animals had normal blood pressure but developed mild dilated cardiomyopathy (increased left ventricle diams. and decreased shortening fraction), which was accompanied by a significant increase in heart rate. Differential gene expression anal. revealed a 2- to 5-fold increase in cardiac expression of atrial natriuretic peptide, **serum**- and glucocorticoid-induced kinase, and early growth response gene 1 as detected by microarrays; renal **serum**- and glucocorticoid-induced kinase was also induced significantly. Altogether,

TG exhibited specific alteration of renal and cardiac functions, thus providing useful pathophysiol. models to gain new insights into the tissue-specific mineralocorticoid signaling pathways.
REFERENCE COUNT: 42 THERE ARE 42 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 18 OF 22 MEDLINE on STN DUPLICATE 10
ACCESSION NUMBER: 2001236194 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11096081
TITLE: Activation of **serum**- and **glucocorticoid -induced protein kinase** (Sgk) by cyclic AMP and insulin.
AUTHOR: Perrotti N; He R A; Phillips S A; Haft C R; Taylor S I
CORPORATE SOURCE: Diabetes Branch, Division of Intramural Research, NIDDK, Bethesda, Maryland 20892, USA.
SOURCE: Journal of biological chemistry, (2001 Mar 23) 276 (12) 9406-12.
Journal code: 2985121R. ISSN: 0021-9258.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200105
ENTRY DATE: Entered STN: 20010517
Last Updated on STN: 20030105
Entered Medline: 20010503
AB Sgk (**serum**- and **glucocorticoid-induced protein kinase**) is a serine/threonine-specific protein kinase that is transcriptionally regulated by **serum**, glucorticoids, and mineralocorticoids. Sgk regulates the amiloride-sensitive sodium channel in kidney principal cells. Insulin and insulin-like growth factor-1 stimulate activity of Sgk by a mechanism mediated by phosphoinositide-dependent kinases (PDK)-1 and -2. In this study, we demonstrate that incubation of transfected cells with 8-(4-chlorophenylthio)-cAMP (8CPT-cAMP; 0.2 mm) led to a 2-fold activation of recombinant Sgk expressed in COS7 cells. Furthermore, the combination of insulin plus 8CPT-cAMP elicited a larger response than either agent alone. The effect of insulin was inhibited by wortmannin (100 nm), but not by the cyclic AMP-dependent protein kinase (PKA) inhibitor, H89 (10 microm). As expected, the effect of 8CPT-cAMP was completely blocked by H89. Surprisingly, the effect of 8CPT-cAMP was also inhibited by wortmannin, suggesting that phosphorylation of Sgk by PDK-1 and/or -2 is required for activation by 8CPT-cAMP. Mutational analysis led to similar conclusions. The Thr(369) --> Ala mutant, lacking the PKA phosphorylation site, was activated by insulin but not 8CPT-cAMP. In contrast, the Ser(422) --> Ala mutant, lacking a PDK-2 phosphorylation site, was inactive and resistant to activation by either insulin or 8CPT-cAMP. In summary, Sgk is subject to complex regulatory mechanisms. In addition to regulation at the level of gene expression, the enzymatic activity of Sgk is regulated by multiple protein kinases, including PKA, PDK-1, and PDK-2. Cross-talk among these signaling pathways may play an important role in the pathogenesis of the hypertension associated with hyperinsulinemia, obesity, and insulin resistance.

L2 ANSWER 19 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN
ACCESSION NUMBER: 2001:527756 HCAPLUS
DOCUMENT NUMBER: 135:238364
TITLE: Phosphoinositide-regulated kinases and phosphoinositide phosphatases
AUTHOR(S): Leslie, Nick R.; Biondi, Ricardo M.; Alessi, Dario R.
CORPORATE SOURCE: MRC Protein Phosphorylation Unit and Division of Signal Transduction Therapy Department of Life Sciences, University of Dundee, Dundee, DD1 5EH, UK
SOURCE: Chemical Reviews (Washington, D. C.) (2001), 101(8), 2365-2380

CODEN: CHREAY; ISSN: 0009-2665
PUBLISHER: American Chemical Society
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 209 refs., focusing on recent research aimed at understanding the mechanism by which activation of phosphoinositide 3-kinase (PI3K), and hence the formation of phosphatidylinositol 3,4,5-trisphosphate [PtdIns(3,4,5)P3], enables 3-phosphoinositide-dependent protein kinase 1 (PDK1) to phosphorylate and activate a group of serine-threonine protein kinases that belong to the AGC subfamily of protein kinases. These AGC family protein kinases include isoforms of protein kinase B (Akt kinase), p70 ribosomal protein S6 kinase (S6K), **serum**- and **glucocorticoid-induced protein kinase** (SGK), and protein kinase C (PKC) isoforms. It is believed that activation of these kinases mediates many of the effects of PI 3-kinase in promoting cell survival and mediating the physiol. responses of cells and tissues to insulin. The importance of phosphoinositide phosphatases PTEN and SHIP, that play key roles in regulating the cellular concn. of PtdIns(3,4,5)P3, and thus regulate the activity of the downstream effector pathways activated by these phosphoinostides, is also discussed.
REFERENCE COUNT: 209 THERE ARE 209 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 20 OF 22 MEDLINE on STN
ACCESSION NUMBER: 2001654900 MEDLINE
DOCUMENT NUMBER: PubMed ID: 11707620
TITLE: Regulation and physiological roles of **serum**- and **glucocorticoid-induced protein kinase** isoforms.
AUTHOR: Lang F; Cohen P
CORPORATE SOURCE: Department of Physiology, University of Tubingen, Germany.. florian.lang@uni-tuebingen.de
SOURCE: Science's STKE [electronic resource] : signal transduction knowledge environment, (2001 Nov 13) 2001 (108) RE17. Ref: 139
Journal code: 100964423. ISSN: 1525-8882.
PUB. COUNTRY: United States
DOCUMENT TYPE: Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 200201
ENTRY DATE: Entered STN: 20011115
Last Updated on STN: 20020420
Entered Medline: 20020114
AB **Serum**- and **glucocorticoid-induced protein kinase** 1 (SGK1) was identified in 1993 as an immediate early gene whose mRNA levels increase dramatically within 30 minutes when cells are exposed to **serum** or glucocorticoids, or both. Subsequently, many other agonists, acting through a variety of signal transduction pathways, have been shown to induce SGK1 gene transcription in cells and tissues. SGK1 is a member of the "AGC" subfamily, which includes protein kinases A, G, and C, and its catalytic domain is most similar to protein kinase B (PKB). Like PKB, SGK1 is activated by phosphorylation in response to signals that stimulate phosphatidylinositol 3-kinase, and this is mediated by 3-phosphoinositide-dependent protein kinase 1 (PDK1) and another protein kinase that has yet to be identified. Thus, SGK1 is remarkable in being activated at both the transcriptional and posttranslational levels by a huge number of extracellular signals. In contrast, little is known about the transcriptional regulation of the two closely related isoforms SGK2 and SGK3, although they can be activated by phosphorylation. The

substrate specificity of SGK isoforms superficially resembles that of PKB in that serine and threonine residues lying in Arg-Xaa-Arg-Xaa-Xaa-Ser/Thr sequences (where Xaa is a variable amino acid) are phosphorylated. However, although they may have some substrates in common, evidence is emerging that SGK1 and PKB phosphorylate distinct proteins and have different functions in vivo. In particular, SGK1 plays an important role in activating certain potassium, sodium, and chloride channels, suggesting an involvement in the regulation of processes such as cell survival, neuronal excitability, and renal sodium excretion. Moreover, sustained high levels of SGK1 protein and activity may contribute to conditions such as hypertension and diabetic nephropathy. This raises the possibility that specific inhibitors of SGK1 may have therapeutic potential for the treatment of several diseases.

L2 ANSWER 21 OF 22 HCAPLUS COPYRIGHT 2005 ACS on STN DUPLICATE 11
ACCESSION NUMBER: 2000:421165 HCAPLUS
DOCUMENT NUMBER: 133:68896
TITLE: Activating **serum** and **glucocorticoid -induced protein kinase** and drug screening
INVENTOR(S): Cohen, Philip; Kobayashi, Takayasu; Deak, Maria
PATENT ASSIGNEE(S): The University of Dundee, UK
SOURCE: PCT Int. Appl., 133 pp.
CODEN: PIXXD2
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.		DATE
WO 2000035946	A1	20000622	WO 1999-GB4232		19991214
W: JP, US					
RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE					
EP 1141003	A1	20011010	EP 1999-961205		19991214
R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI					
JP 2002533063	T2	20021008	JP 2000-588203		19991214
PRIORITY APPLN. INFO.:			US 1998-112217P	P	19981214
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AB A method of activating **serum** and **glucocorticoid-induced protein kinase** (SGK) is provided wherein the SGK is phosphorylated. The SGK may be phosphorylated by PDK1 and/or a prepn. contg. PDK2 activity. A method of identifying a compd. that modulates the activity of SGK is provided, wherein the activity of SGK is measured by measuring the phosphorylation by SGK of a polypeptide comprising an amino acid sequence corresponding to the consensus sequence (Arg/Lys; preferably Arg)-X-(X/Arg)-X-X-(Ser/Thr)-Z wherein X indicates any amino acid, X/Arg indicates any amino acid, with a preference for arginine, and Z indicates that the amino acid residue is preferably a hydrophobic residue. The SGK may be activated by phosphorylation. The invention relates to screening methods for finding new drugs or lead compds.
REFERENCE COUNT: 3 THERE ARE 3 CITED REFERENCES AVAILABLE FOR THIS RECORD. ALL CITATIONS AVAILABLE IN THE RE FORMAT

L2 ANSWER 22 OF 22 MEDLINE on STN DUPLICATE 12
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TITLE: Characterization of the structure and regulation of two novel isoforms of **serum**- and **glucocorticoid-induced protein kinase**.

AUTHOR: Kobayashi T; Deak M; Morrice N; Cohen P
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AB The catalytic domain of **serum**- and **glucocorticoid-induced protein kinase** (SGK) is 54% identical with protein kinase B (PKB) and, like PKB, is activated in vitro by 3-phosphoinositide-dependent protein kinase-1 (PDK1) and in vivo in response to signals that activate phosphatidylinositol (PI) 3-kinase. Here we identify two novel isoforms of SGK, termed SGK2 and SGK3, whose catalytic domains share 80% amino acid sequence identity with each other and with SGK (renamed SGK1). Like SGK1, the mRNA encoding SGK3 is expressed in all tissues examined, but SGK2 mRNA is only present at significant levels in liver, kidney and pancreas and, at lower levels, in the brain. The levels of SGK2 mRNA in H4IIE cells and SGK3 mRNA in Rat2 fibroblasts are not increased by stimulation with **serum** or dexamethasone, whereas the level of SGK1 mRNA is increased greatly. SGK2 and SGK3 are activated in vitro by PDK1, albeit more slowly than SGK1, and their activation is accompanied by the phosphorylation of Thr(193) and Thr(253) respectively, the residues equivalent to the Thr in the 'activation loop' of PKB that is targeted by PDK1. The PDK1-catalysed phosphorylation and activation of SGK2 and SGK3, like SGK1, is greatly potentiated by mutating Ser(356) and Ser(419) respectively to Asp, these residues being equivalent to the C-terminal phosphorylation site of PKB. Like SGK1, SGK2 and SGK3 are activated 5-fold via a phosphorylation mechanism when cells are exposed to H(2)O(2) but, in contrast with SGK1, activation is only suppressed partially by inhibitors of PI 3-kinase. SGK2 and SGK3 are activated to a smaller extent by insulin-like growth factor-1 (2-fold) than SGK1 (5-fold). Like PKB and SGK1, SGK2 and SGK3 preferentially phosphorylate Ser and Thr residues that lie in Arg-Xaa-Arg-Xaa-Xaa-Ser/Thr motifs.

=> log y

COST IN U.S. DOLLARS	SINCE FILE ENTRY	TOTAL SESSION
FULL ESTIMATED COST	40.69	40.90

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)	SINCE FILE ENTRY	TOTAL SESSION
CA SUBSCRIBER PRICE	-5.84	-5.84

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